Socioeconomic disparities in children's cognitive development: Longitudinal dynamics and stress mechanisms

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Eidesstattliche Erklärung

Hiermit erkläre ich an Eides statt,

- dass ich die vorliegende Arbeit selbstständig und ohne unerlaubte Hilfe verfasst habe,
- dass ich mich nicht bereits anderwärts um einen Doktorgrad beworben habe und keinen Doktorgrad in dem Promotionsfach Psychologie besitze, und
- dass ich die zugrunde liegende Promotionsordnung vom 3. August 2006 kenne.

Berlin, den 18.12.2017

Laurel Raffington

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Summary

Between–person differences in cognitive development in childhood vary along gradients of socioeconomic status (SES). By preschool age, lower SES children perform nearly one standard deviation below higher SES children. Yet, research has largely ignored longitudinal within–person dynamics, despite evidence that SES, especially income, also changes over time. Delineating within–person dynamics strengthen inferences about bivariate relationships. Furthermore, fairly little is known about the mechanisms by which SES explains cognitive disparities. Lower SES environments are marked by high chronic stress exposure, which has led to the hypothesis that dysregulation of the stress response system resulting in altered cortisol secretion mediates disparities in cognition. However, research on cortisol secretion in childhood has been characterized by severe methodological limitations, which may partly explain mixed results associating SES–related stressors with both higher and lower basal cortisol levels. Additionally, childhood cognition is closely linked with decision–making, psychopathology, and physical health throughout the lifespan, which makes it a candidate modifier of the effects of stress exposure on cortisol secretion. Lastly, evidence across multiple systems linking SES–related disparities to children's cognitive functioning via altered cortisol secretion and neural structure is absent.

Based on these considerations, this dissertation explores longitudinal within–person dynamics of income and cognition and investigates stress–related mechanisms involved in SES–related disparities of cognitive functioning in child development. The dissertation consists of four research aims explored in three publication–oriented empirical papers. At the time of printing, *Paper I* is in the second revision, Paper *II* is resubmitted after the first revision, and *Paper III* is under review.

First, *Paper I* explores longitudinal dynamics between income and cognitive development through middle childhood and early adolescence (4–12 years). Bivariate dual change score models provide evidence that within–person income gains and losses continue to predict poor children's cognitive performance in later childhood. This effect was not found in children who never lived below the poverty threshold. Interestingly, there was also a novel effect in which child cognitive scores positively predict income change of the parents, suggesting bidirectional income–cognition dynamics.

Second, *Paper II* and *III* apply advanced statistical methods to model multiple cortisol measurements investigating whether higher stress and lower income are associated with cortisol secretion in middle childhood. In *Paper II*, hierarchical mixed effects models revealed that higher parenting stress of parents was associated with lower total diurnal cortisol level including the diurnal cortisol awakening response (CAR) in a sample of 6–to–7–year–old children. In *Paper III*, latent–change structural equation models captured the CAR and cortisol stress reactivity (CSR) in response to acute laboratory stress in a separate sample of 6–to–7–year–old children. Lower family income was associated with blunted cortisol reactivity in both CAR and CSR.

Third, *Paper II* investigates whether cognitive control moderates effects of stress on cortisol secretion. Indeed, at lower levels of child cognitive control higher parenting stress was associated with lower cortisol levels, whereas higher cognitive control ability dissociated parenting stress and cortisol secretion. Fourth, *Paper III* also tests the hypothesis that altered dynamic cortisol secretion is negatively related to hippocampal–dependent memory functioning and hippocampal volume. We found cortisol hyporeactivity to stress was associated with lower memory among lower–income children, who also had smaller hippocampal volumes.

Taken together, this dissertation provides longitudinal evidence for the notion that family income gains and losses predict poor children's cognitive development throughout later childhood (*Paper I*). Remarkably, children's cognition also predicts their parents' earning ability, indicating previously neglected bidirectional family dynamics. Moreover, this dissertation provides cross–sectional evidence for a stress mechanism involved in SES disparities of cognitive functioning. Both higher stress and lower income are associated with patterns of hypocortisolism in middle childhood (*Paper II & III*). Further, cognitive control skills may buffer against the embedding of stress exposure in diurnal cortisol secretion (*Paper II*). Lastly, cortisol hyporeactivity to stress found in low–income children is uniquely associated with their lower memory performance. Notably, this dissertation does not warrant environmentally– or genetically–mediated assumptions of causality. Future intervention studies should continue to explore whether facilitating family income gains, reducing stress exposure, normalizing cortisol secretion, or improving children's stress coping by training cognitive control alleviates SES–related disparities in cognition.

Zusammenfassung

Individuelle Unterschiede in der kognitiven Entwicklung von Kindern korrelieren mit ihrem sozioökonomischen Status (*engl. socioeconomic status*, SES). Kinder mit niedrigerem SES zeigen bereits im Vorschulalter fast eine Standardabweichung niedrigere Schulleistung als reichere Kinder. Allerdings hat die bisherige Forschung längsschnittliche, intra–individuelle Dynamiken ignoriert, obwohl SES–Indikatoren, vor allem das Familieneinkommen, sich durchaus über die Zeit hinweg verändern können. Dabei erlaubt die Analyse von intra–individuellen Dynamiken ein besseres Verständnis dafür, ob und wie das Familieneinkommen die kognitive Entwicklung von Kindern vorhersagt. Des Weiteren sind die Mechanismen, die zu SES–bedingten Diskrepanzen in der kognitiven Entwicklung führen, weitgehend unbekannt. Gemäß einer Hypothese ist es die Dysregulation der Sekretion des Stresshormons Cortisol die SES Unterschiede vermittelt, da niedriger SES robust mit höherem chronischen Stress assoziiert. Allerdings ist die Erforschung der Cortisolsekretion in Kindern durch methodische Einschränkungen gekennzeichnet, die möglicherweise widersprüchliche Forschungsergebnisse, die niedrigeren SES teils mit höheren und teils niedrigeren basalen Cortisolwerten assoziieren, erklären. Zudem sind kognitive Kontrollfunktionen von Kindern ihrerseits mit individuellen Unterschieden in der Entscheidungsfindung, psychischen und physischen Gesundheit assoziiert. Dadurch könnte kognitive Kontrolle die Einbettung von chronischem Stress in das Stresssystem moderieren. Letztlich fehlt empirische Evidenz, dass individuelle Unterschiede in Kognition im Zusammenhang mit SES über eine Cortisol–Dysregulation und veränderte neuronale Strukturen vermittelt werden. Die vorliegende Dissertation leistet einen Beitrag zum besseren Verständnis der intra–individuellen Interaktion von Einkommen und kognitiver Entwicklung und untersucht mögliche Stressmechanismen als Mediatoren von SES–bedingten Unterschieden in der kognitiven Funktionsfähigkeit. Die Dissertation ist publikationsorientiert und besteht aus vier Forschungsschwerpunkten, die in drei Studien behandelt werden. Zum Zeitpunkt der Dissertationsabgabe ist *Studie I* in der zweiten Revision, *Studie II* ist nach der ersten Überarbeitung wieder eingereicht und *Studie III* ist eingereicht.

Erstens untersucht *Studie I* längsschnittliche Dynamiken zwischen dem Familieneinkommen und der kognitiven Entwicklung in der späteren Kindheit (4–12 Jahre). Strukturgleichungsmodelle zeigen, dass intra–individuelle Zugewinne und Verluste im Einkommen die kognitive Entwicklung von armen Kindern vorhersagen. Interessanterweise dokumentieren wir auch, dass der kognitive Stand der Kinder umgekehrt auch Veränderungen im Elterneinkommen beeinflusst.

Zweitens wenden *Studie II* und *III* fortgeschrittene statistische Methoden an, um Cortisolmessungen mit höherer Reliabilität zu modellieren. So erkunden wir, ob höherer Stress und ein niedrigeres Einkommen mit Cortisol–Dysregulation im mittleren Kindesalter verknüpft sind. *Studie II* zeigt, dass höherer Stress der Eltern in Bezug auf ihre Elternrolle mit geringerer Gesamtausschüttung von Cortisol über den Tag bei ihren 6–bis–7–jährigen Kindern assoziiert ist. *Studie III* erweitert diese Befunde in einer separaten Stichprobe von 6–bis–7–jährigen Kindern und weist auf, dass ein geringeres Familieneinkommen mit niedrigeren Cortisol–Ausschüttungen am Morgen sowie in der Reaktion auf akuten Stress assoziiert ist. Drittens zeigt *Studie II*, dass nur Kinder mit geringerer kognitiver Kontrolle auch verringerte Cortisol–Sekretion aufweisen, wenn ihre Eltern höheren Stress berichten. Viertens ist reduzierte Cortisolausschüttung in Reaktion auf akuten Stress mit niedrigeren Gedächtnisleistungen bei Kindern mit geringerem Familieneinkommen verbunden. Diese weisen auch ein kleineres Hippokampus–Volumen auf (*Studie III)*.

Die vorliegende Dissertation weist im Längsschnitt nach, dass Veränderungen im Familieneinkommen die kognitive Entwicklung von armen Kindern im späteren Kindesalter vorhersagen (*Studie I*). Bemerkenswerterweise wirkt kindliche Kognition auch auf Veränderungen im elterlichen Einkommen, was auf bisher vernachlässigte bidirektionale Familiendynamiken hinweist. Zudem liefert die Dissertation querschnittliche empirische Evidenz für die Hypothese, dass ein stressbezogener Mechanismus in SES–bedingten Unterschieden kognitiver Entwicklung involviert ist. Höherer Stress und ein geringeres Einkommen waren beide mit Anzeichen des Hypocortisolismus in Kindern charakterisiert (*Studie II* und *III*). Außerdem gab es Hinweise, dass kognitive Kontrolle vor den Effekten von Stress schützen könnte (*Studie II*). Schließlich zeigt die Dissertation erstmalig, dass hyporeaktive Cortisolprofile in Reaktion auf akuten Stress in ärmeren Kindern mit deren niedrigerer Gedächtnisleistung assoziiert ist (*Studie III*). Beachtenswert ist, dass Ergebnisse dieser Dissertation nicht auf Umwelt oder genetisch vermittelte Kausalität schließen lassen. In zukünftigen Interventionsstudien sollte weiter untersucht werden, ob Interventionen die Einkommensgewinne stärken, Stress reduzieren oder die Reaktion von Kindern auf akuten Stress verbessern, womöglich vermittelt über eine Stärkung von kognitiven Kontrollfähigkeiten, kognitive Unterschiede im Zusammenhang mit SES verringern.

List of original papers

Paper I

Raffington, L., Prindle, J. J., & Shing, Y. L. (2018). Income gains predict cognitive functioning longitudinally throughout later childhood in poor children. *Developmental Psychology*. Advance online publication. http://dx.doi.org/10.1037/dev0000529

Paper II

Raffington, L., Schmiedek, F., Heim, C., & Shing, Y. L. (2018). Cognitive control moderates parenting stress effects on children's diurnal cortisol. *PLoS ONE, 13*(1): e0191215. http://doi.org/10.1371/journal.pone.0191215

Paper III

Raffington, L., Prindle, J., Keresztes, A., Binder, J., Heim, C. & Shing, Y. L. (2018). Blunted cortisol stress reactivity in low-income children relates to lower memory function. *Psychoneuroendocrinology, 90,* 110–121. http://doi.org/10.1016/j.psyneuen.2018.02.002

List of abbreviations

ANS	Autonomic nervous system
CAR	Cortisol awakening response
CSR	Cortisol stress reactivity
DCSM	Dual–change score model
GC	Glucocorticoid
GR	Glucocorticoid receptor
HPA	Hypothalamus–pituitary–adrenal
MRI	Magnetic resonance imaging
PFC	Prefrontal cortex
SD	Standard deviation
SEM	Structural equation model
SES	Socioeconomic status
TSST–C	Trier Social Stress Test for Children

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1. Introduction

A large literature documents the correlation of between–person differences in cognitive development and socioeconomic status (SES). SES is most commonly indicated by family income, parental education, and occupation and has been utilized to capture what can be described as environmental circumstances of those 'better off' and 'worse off' in society (Farah, 2017). In addition, a substantial proportion of the SES–cognition association is genetically–mediated (Spinath & Bleidorn, 2017). Studies suggest that SES–related disparities are found early in infancy (Betancourt et al., 2016; Tomalski et al., 2013), widen over age (Carneiro & Heckman, 2003; von Stumm & Plomin, 2015), and are most pronounced in poverty (Dearing, McCartney, & Taylor, 2001). Nevertheless, longitudinal bidirectional dynamics have remained unexplored. Furthermore, very little is known about the mechanisms involved in associating lower SES wth lower cognitive performance. Since low SES environments are marked by high chronic stress exposure, one of the hypothesized mechanisms is that stress response system dysregulation that is measurable in altered cortisol secretion mediates disparities in cognition by effects of cortisol on brain circuits (Lupien, McEwen, Gunnar, & Heim, 2009). Yet, research on SES–related stress and children's cortisol secretion is characterized by severe methodological limitations and paints a mixed picture of altered cortisol secretion. Furthermore, childhood cognition is closely linked with decision–making, psychopathology, and physical health throughout the lifespan and may therefore modify the association of stress exposure and cortisol secretion. Lastly, links between multiple systems from SES to cognition via altered cortisol secretion and aberrant neural structure are unexplored. Hence, empirical evidence for the notion that cortisol secretion mediates disparities in cognition is lacking.

In my dissertation, I investigate longitudinal dynamics of change between income and cognition, allowing for moderation of poverty. Second, I contribute to emerging research on mechanistic pathways associating SES and stress to individual differences in endocrine, neural, and cognitive functioning. In section 2 I will first outline the theoretical and empirical background of my dissertation reviewing research on the development of multiple systems. I will then give a short summary of each of the papers included in this dissertation. Finally, I discuss the results of my studies, considering limitations and future research directions.

2. Theoretical and empirical foundations

In the following sections, I first briefly exemplify core concepts of cognitive development and its neural correlates that inform subsequently examined research on individual differences along SES gradients. I then turn to empirical evidence on the main mechanisms assumed to explain SES–related cognitive disparities. These lead to a discussion on how to operationalize SES. Focusing on mechanisms related to stress, I review empirical studies on cortisol secretion in the developmental literature and highlight methodological limitations. Lastly, I summarize emerging research on SES–related stress, cortisol secretion, and cognition, which leads me to the aims of my dissertation.

2.1 Cognitive and neural development in childhood

Childhood is marked by rapid development in neural structure that is complexly coupled to the cognitive functions these structures correlate with. As a first example, the medial temporal lobe including the hippocampus develops speedily in infancy and early childhood (0–5 years) supporting relatively mature associative memory skills by middle childhood (5–10 years) (Giedd et al., 1999; Keresztes et al., 2017; Menon, Boyett–Anderson, & Reiss, 2005; Shing et al., 2010; Shing, Werkle–Bergner, Li, & Lindenberger, 2008; Sowell et al., 2003). The hippocampus is a multi–functional region that, for example, is involved in the acquisition of new relational information during memory encoding and reinstatement of memory representations during retrieval (for review, see Shing et al., 2010). Furthermore, the hippocampus is a highly plastic region that continually adjusts to the environment throughout the lifespan, reflecting lifelong learning (Eriksson et al., 1998; Freund et al., 2013). Secondly, the prefrontal cortex (PFC) undergoes an enormous amount of synaptic growth and development in the first two years of life, at 7 to 9 years of age, and again around 15 years of age, but does not reach maturity until much later in young adulthood (Huttenlocher, 2009; Lenroot & Giedd, 2006; Thatcher, 1991). Correspondingly, children show rapid improvements in executive function abilities throughout early childhood years (Davidson, Amso, Anderson, & Diamond, 2006) and continue improving at a slower rate into young adulthood (De Luca & Leventer, 2010), which is reflected in the development of the PFC and, more specifically, networks of fronto–parietal and cingulo–opercular brain regions (Dosenbach, Fair, Cohen, Schlaggar, & Petersen, 2008; Luna, Padmanabhan, & O'Hearn, 2010). The PFC is critically involved in executive functions, including for example the maintenance of focused attention and voluntary inhibition of motor and sensory regions (for review, see Luna, Padmanabhan, & O'Hearn, 2010). Similarly, strategic control aspects of memory functioning show protracted development (Shing et al., 2010, 2008). More so, the plasticity of the hippocampus and extended development of the PFC make these brain areas especially amenable to environmental influence (Lupien et al., 2009). Thus, cognitive and neural development is not an unfolding of genetic and maturational processes, but rather presents an experience–dependent

(i.e. functional neural development relies on experiential input) and experience–expectant (i.e. neural regions are tuned to incorporate experiential input) interaction with the environments children inhabit and explore (Fox, Levitt, & Nelson III, 2010; Rinaldi & Karmiloff–Smith, 2017; Sweatt, 2009). Critically, genetic and environmental influences on cognition correlate and interact, and their relative contribution to phenotypic profiles varies over ontogeny (Scarr & McCartney, 1983; Spinath & Bleidorn, 2017). Heritability studies estimate that environmental influences are most pronounced early in childhood and additive genetic variation accounts for up to 70% of the variation in cognition by adolescence (Tucker–Drob & Briley, 2014) (see 2.3 for further discussion on environmental and genetic effects).

2.2 SES–related disparities in cognitive and neural development

A robust finding in developmental psychology is that between–person differences in cognitive and academic performance varies as a function of family SES. In cross–sectional studies, achievement tests of reading and math as well as cognitive tests of language, executive functions, and memory show strong to moderate linear associations with SES with gaps of up to one standard deviation (SD) between poor and rich (Duncan & Magnuson, 2011; Farah et al., 2008; Lawson, Hook, & Farah, 2017; Montroy, Bowles, Skibbe, McClelland, & Morrison, 2016; Noble, McCandliss, & Farah, 2007). Longitudinal research investigating SES and cognitive development across childhood has recently accumulated. Studies show that children of lower SES have lower initial levels and slopes of intelligence (von Stumm & Plomin, 2015), executive functions, and self–regulation (Hackman, Gallop, Evans, & Farah, 2015; Montroy et al., 2016), as well as verbal comprehension and math ability (Crosnoe, Leventhal, Wirth, Pierce, & Pianta, 2010; Hair, Hanson, Wolfe, & Pollak, 2015; National Institute of Child Health and Human Development Early Child Care Research Network, 2005; Wang et al., 2017). The disparities in math ability are partially mediated by executive functions longitudinally (Lawson & Farah, 2017). Additional cross–sectional and longitudinal evidence is newly amassing to suggest that SES differences in cognitive functions are found in the brain structures that support them. Lower SES has been related to smaller hippocampal volume (Brody et al., 2017; Hair et al., 2015; Hanson, Chandra, Wolfe, & Pollak, 2011; Jednoróg et al., 2012; Luby et al., 2013; Noble et al., 2015; Noble, Grieve, et al., 2012; Noble, Houston, Kan, & Sowell, 2012; Rao et al., 2010; Yu et al., 2017) and smaller cortical volume including the PFC (Hair et al., 2015; Hanson et al., 2012) or decreased cortical surface areas including the PFC (Noble et al., 2015). Considerably less attention has been paid to white matter structure, although evidence for differences in fronto–limbic white matter tracts involved in cognitive and emotional functions (uncinate fasciculus and cingulum bundle) and association fibers (inferior longitudinal fasciculus, superior longitudinal fasciculus) exists (Chiang et al., 2011; Dufford & Kim, 2017; Jednoróg et al., 2012; Luby et al., 2013). Additionally, neuroscientific evidence suggests SES–related disparities in gray matter volume

are found at ages as early as 1 month in healthy infants (Betancourt et al., 2016), and in functional brain activity measured with EEG in 6–month–olds (Tomalski et al., 2013). Although SES–related disparities in EEG power may not present themselves shortly after birth (Brito, Fifer, Myers, Elliott, & Noble, 2016), the association with gestational age, birth weight, and prenatal stress suggests that disparities are likely to exist before birth even if they may not appear until later (Aizer & Curley, 2014). Furthermore, cognitive (Carneiro & Heckman, 2003; Cunha, Heckman, Lochner, & Masterov, 2006; Hanson et al., 2013; Moffitt et al., 2011; von Stumm & Plomin, 2015) and neural (Hanson et al., 2013) disparities substantially widen across earlier childhood, and rank–order stability of cognitive performance is very high by the end of the first decade of life (Hackman et al., 2014; Tucker–Drob & Briley, 2014). Collectively, these studies suggest that between–person SES–related disparities in neural and cognitive functioning are found early in ontogenetic development and widen over early and middle childhood, reaching high levels of stability in between–person rank–order by later childhood.

Importantly, both cross–sectional and longitudinal SES research has been almost exclusively genetically uninformed and correlational (Duncan, Magnuson, & Votruba–Drzal, 2017) and thus do not provide evidence for environmental causation. SES indicators have been treated as static predictors of child development (Crosnoe et al., 2010; Lawson & Farah, 2017; von Stumm & Plomin, 2015; Wang et al., 2017), although these indicators, especially income, also change over time (Duncan, Ziol–Guest, & Kalil, 2010). Delineating repeated, time–lagged measurements to explore change, for example with structural equation models (SEMs), strengthens inferences on bivariate relationships (Adler & Rehkopf, 2008; Hamaker, Kuiper, & Grasman, 2015). For instance, change in income–to–needs predicts child cognition at age 3 for poor, but not for families who were never poor (Dearing et al., 2001; Mistry, Biesanz, Taylor, Burchinal, & Cox, 2004). In addition, quasi–experimental studies suggest that income received when a child is young (ages 0–5 years) has stronger lasting impacts on cognitive and school achievement than does income received during later childhood or adolescence (Duncan, Brooks–Gunn, & Klebanov, 1994; Duncan, Yeung, Brooks–Gunn, & Smith, 1998; Heckman, 2006). Therefore, previous research has shown that changes in income during early childhood predict child cognition for families living in poverty. It is less established whether family income changes in later childhood and adolescence continue to predict cognitive functioning. Furthermore, although cognitive functioning and neural structure is associated with SES across the whole SES continuum in a gradient manner, the gradient seems to be steeper at the lower end, which could signal some threshold effects (Hair, Hanson, Wolfe, & Pollak, 2015; Noble et al., 2015). Thus, contrasting income–cognition dynamics along large SES variation and within poverty informs gradient versus threshold relationships.

In addition to largely neglecting the within–person dynamic relationship of income on cognition, this literature has ignored potential effects of children on their parents' ability to earn income. However, given evocative and bidirectional effects between children's behaviors and their

parents' parenting styles and psychological well–being as well as their shared genetic profiles (Bradley & Corwyn, 2013; Miner & Clarke–Stewart, 2008; Pike, McGuire, Hetherington, Reiss, & Plomin, 1996; Scarr & McCartney, 1983; Tucker–Drob & Harden, 2012a), it is possible that children also influence family income. The dynamic bidirectional relationship between family income and child cognition over middle childhood and early adolescence is yet to be explored.

2.3 Mediators of SES–related disparities

Theories of social causation propose two main environmental pathways as mediators of the association of SES and child cognition. First, the enrichment pathway, also called the family investment model, describes dissimilarities in enriching and cognitively stimulating experiences along SES (Hackman, Farah, & Meaney, 2010). This includes a reduced occurrence of growth fostering materials and experiences, (e.g., stimulating toys or music classes), provision for active stimulation (e.g., reading) and family participation in developmentally stimulating experiences (e.g., outings) in lower SES families (Farah et al., 2008; Totsika & Sylva, 2004). Parental expenditures on enriching activities for their children between upper and lower SES has substantially increased in recent years, which could partly explain increasing gaps in children's cognition (Duncan & Murnane, 2011; Raver, Roy, & Pressler, 2015).

Second, there is robust evidence that lower SES families experience higher stress, which can be differentiated into stressor exposure, stress perception and stress response (Lupien, King, Meaney, & McEwen, 2001). Lower SES coincides with higher rates of stressor exposure, including more individual stressful life events (Dohrenwend, 1973), and chronically chaotic and dangerous homes and neighborhoods with higher rates of interpersonal conflict (Bradley & Corwyn, 2002; Evans, 2004; Hackman et al., 2010). Subsequently, stressor exposure may or may not lead to stress perception, which describes a psychological state in which an individual feels inadequately equipped to cope with the demands of the environment (Adler et al., 1994). Lower SES also robustly and strongly correlates with stress perception, including perceived social class discrimination (Fuller–Rowell, Evans, & Ong, 2012), and parenting stress (Gershoff, Aber, Raver, & Lennon, 2007; Hackman et al., 2015). Stress may affect parenting behaviors that could create stress for the child (Evans, 2004; Finegood & Blair, 2017; McLoyd, 1990; Murgatroyd & Nephew, 2013). Additionally, experimental evidence suggests that parents remain co–regulators of stress responses to acute challenges into middle childhood (Hostinar, Johnson, & Gunnar, 2015b) and parent–child interactions can elicit a stress response dependent on the relationship quality (Gunnar, Talge, & Herrera, 2009). However, a more valid measure of children's stress experiences would be to evaluate children's self–reported stress perception. Yet, the lack of correlation between parental and child stress reports may imply that young children are not reliable self–reporters (Bögels, van Oosten, Muris, & Smulders, 2001; Caster, Inderbitzen, & Hope, 1999; Tein, Roosa, & Michaels, 1994; Weissman, Orvaschel, & Padian, 1980),

in contrast to children over 9 years (LeMoult, Ordaz, Kircanski, Singh, & Gotlib, 2015; Maldonado et al., 2008; Wolf, Nicholls, & Chen, 2008). Nonetheless, even among high–risk, low–income families, stress perception varies considerably, such that they can be clustered into subgroups of high, intermediate, and low stress perception separated by up to 2 SDs (Hustedt, Vu, Bargreen, Hallam, & Han, 2017). Third, perceiving stress initiates a physiological cascade in the body, which is referred to as the stress response. While robust empirical evidence shows lower SES is marked by higher stressor exposure and stress perception, emerging evidence on children's stress response is mixed (as reviewed in section 2.6).

Furthermore, several environmental circumstances and stressors that covary to some degree with SES are typically not studied as mediators of the SES concept, but treated as confounders to control for or are discussed separately. This includes maltreatment and trauma (Evans & Kim, 2010; Häuser, Schmutzer, Brähler, & Glaesmer, 2011; Moog et al., 2018), developmental and health disorders, prematurity, exposure to environmental toxins and nutritional deficiencies as well as racism (Williams, Mohammed, Leavell, & Collins, 2010). These literatures in turn include SES as a control variable. Crucially, many of these chronic stressors, for example maltreatment (Lawson, Camins, et al., 2017) and racism (Noble et al., 2015; Sirin, 2005), have been shown to have distinguishable effects from SES on both a neural and cognitive level. Nevertheless, these variables are usually controlled for by exclusion criteria (e.g., reported trauma, developmental and health disorders, prematurity) or by statistically controlling them (e.g., race, ethnicity, and immigration). However, this reduces external validity because the prevalence rates are higher in lower SES (Adler et al., 1994; Bradley & Corwyn, 2002; Häuser et al., 2011) and systematically underestimates outcomes of SES–related disadvantage.

Critically, the correlation of SES, environmental quality, and cognition does not preclude genetically–mediated effects in studies that are not genetically informed (Ericsson et al., 2017; Krapohl & Plomin, 2016; Polderman et al., 2015; Spinath & Bleidorn, 2017). In contrast to social causation theories, social selection and interactionist models highlight how individual characteristics influence SES–cognition correlations (Conger & Donnellan, 2007). For example, parents may provide their children with a lower SES childhood environment and transmit genes predisposing them to lower cognitive performance. Behavior genetic twin studies and molecular genetic research has provided empirical evidence that more proximal phenotypic traits like brain development (Shonkoff et al., 2012), cognition (Engelhardt, Briley, Mann, Harden, & Tucker–Drob, 2015), and distal outcomes such as SES (Hill et al., 2016; Selzam et al., 2017) as well as their correlation are genetically influenced to a substantial degree (Ericsson et al., 2017; Krapohl & Plomin, 2016; Spinath & Bleidorn, 2017). For example, Ericsson et al. (2017) show that childhood SES correlations with between–person differences in cognitive ability and within–person cognitive change in old age are entirely explained by genetic effects when comparing monozygotic and dizygotic twins reared together or apart. This suggests that SES variation is not orthogonal, but partially aligned with genetic

variation in cognition. On the other hand, longitudinal mediation (Hackman et al., 2015), adoption studies (Capron & Duyme, 1989; Kendler, Turkheimer, Ohlsson, Sundquist, & Sundquist, 2015; van IJzendoorn, Juffer, & Poelhuis, 2005), (quasi–)experimental and intervention studies (Costello, Compton, Keeler, & Angold, 2003; Duncan et al., 1998; Heckman, 2006) and genetically informed studies (Tucker-Drob & Briley, 2014; Tucker-Drob, Briley, & Harden, 2013; Tucker-Drob & Harden, 2012b) provide evidence that environments along SES strata are also likely to play a causal role in childhood cognitive development. Thus, cognitive disparities along the SES gradient are explained by both environmentally– and genetically–mediated effects, which are known to interact and correlate in ways we do not yet fully comprehend (Scarr & McCartney, 1983).

2.4 Measurement of SES–related effects

There is ongoing debate about how to operationalize SES, because the correlation of SES indicators amongst each other is far from perfect. Some argue for the use of composite scores, that is, based on a factor analysis of income, education, and occupation (Duncan & Magnuson, 2012) Others propose looking at SES indicators separately, since maternal education may more directly relate to parenting and parent–child interaction, whereas income may be more closely linked to material resources (Duncan & Magnuson, 2012; Noble et al., 2015). Similarly, parental income and education may have differential effects on cognitive (Farah et al., 2008) and neural outcomes (Noble et al., 2015). This debate is further complicated by the fact that the correlation of these predictors depends on the degree of SES variance sampled in individual studies (Betancourt et al., 2016) and, presumably, cross–cultural differences in the severity of social disparities and what access they allow (e.g., universal healthcare provision in Germany vs. restricted access in the US). Farah (2017) proposes reporting effects of all SES indicators and their composite, but prioritizing the main indicator of interest in the results. Although this would aid comparability across studies, it also dramatically increases the number of statistical comparisons. Similarly, experimenter–rated measures of the environment and parent–reported questionnaires are best at distinguishing between minimally and maximally stimulating environments, but are limited in measuring moderate and maximal quality environments (Votruba–Drzal, 2003). A combination of gradient and threshold effects may reflect differently weighted combinations of causes operating at different levels of SES (Farah, 2017). Therefore, environmental measures may not necessarily be more valid alternatives to SES indicators.

These issues illustrate that SES is not a ‘natural kind’ (Quine, 1969), but a complex abstraction of environmental circumstances with unresolved measurement issues, poor definitions of SES (Braveman et al., 2005), and a lack of experimental control. SES is therefore an umbrella term for the myriad of environmental circumstances that are unequally distributed across populations – be they genetic or environmental in origin. Yet, given the magnitude of cognitive differences along SES strata, the SES context cannot just be ignored if we seek to understand between–person differences. I

take the view that operationalizing SES or SES–related environmental measures should be a sample–specific decision that is dependent on the research question. In large samples that mirror the sociodemographic configuration of the population, composite SES scores may capture the breadth of variance in environmental quality. While such samples are the most desirable, they are not always obtainable by researchers. In contrast, in severely impoverished samples, it may be more informative to use indicators of, say, stress perception, which may mark between–person differences more than income and education (Hustedt et al., 2017). In samples of more moderately varying SES, associations with child cognition are still found (Obradović, Portilla, & Ballard, 2016), but income and education are not as highly correlated and may have diverging associations with child outcomes (Farah et al., 2008; Noble et al., 2015). Those samples may experience different levels of stress related to financial strain (Hustedt et al., 2017; Raver et al., 2015) or other factors correlated with SES (Bradley & Corwyn, 2002), despite being relatively well–educated in socially recognized occupations. Furthermore, income is the most variable SES indicator (Duncan, Ziol–Guest, & Kalil, 2010) and most commonly targeted by policy programs. Thus exploring effects of income may be of specific interest to researchers. Therefore, I rather indiscriminately review studies operationalizing SES in different ways or using environmental measures of stress or enrichment as predictors, whilst acknowledging that operationalizing SES such that it facilitates comparison across studies and disentangles environmental and genetic pathways is an important task for this field.

2.5 Salivary cortisol concentrations in states of stress

Research investigating the role of stress in relation to SES has sought a reliable biomarker to better understand 'how stress gets under the skin' (Lupien et al., 2001; McEwen, 2012). This is especially important in the developmental literature, because young children may be unreliable reporters of their own stress experiences (Bögels et al., 2001; Caster et al., 1999; Tein et al., 1994; Weissman et al., 1980) and the developing brain is disproportionally more vulnerable to the adverse effects of stress than the adult brain (Knudsen, Heckman, Cameron, & Shonkoff, 2006). Rodent and non–human primate research has shown that glucocorticoid (GC) steroid hormones are causally affected by stress exposure, showing patterns of GC hyperresponsive or hyporesponsive dysregulation, depending on the type and frequency of the stressor (e.g., repeated maternal separation, social isolation, or human handling), and age of stressor exposure (Levine, 2006; Lupien et al., 2009; Meaney, 2001; Sánchez, Ladd, & Plotsky, 2001). Furthermore, sustained increases in stressor exposure, GC secretion, and administration render brain regions rich in GC receptors (GRs), like the hippocampus and PFC, vulnerable to adverse neuroplastic changes, reducing spine density, synaptogenesis, excitability, and dendritic atrophy that reduce neural volume and impair cognition (Lupien et al., 2009; McEwen, 2000; Popoli, Yan, McEwen, & Sanacora, 2012).

Non–pharmacological research exploring mechanisms of stress system functioning in humans has examined two different temporal dimensions of salivary cortisol output, which is the main GC in humans and is regulated by the hypothalamic–pituitary–adrenal (HPA) axis. First, HPA axis reactivity to stress can be thought of as the antecedent of energy mobilization, immune suppression, and feedback inhibition, which is beneficial in the short–term by enabling the organism to deal with challenges, but comes at a cost with long–term activation (Danese & McEwen, 2012). Notably, the stress response is not limited to HPA axis activity, but involves the autonomic nervous system (ANS) and neurotransmitter systems, such as oxytocin, dopamine, and serotonin (LaPrairie, Heim, & Nemeroff, 2010). These systems are not discussed here for reasons of space restriction. Second, cortisol secretion has a pronounced diurnal rhythm that can be divided into two relatively discrete components characterized by peak levels following morning awakening (the cortisol awakening response, CAR) and declining levels thereafter (diurnal slope) (Clow, Hucklebridge, Stalder, Evans, & Thorn, 2010).

Both cortisol levels in response to acute stress and diurnal secretion have been extensively studied as biomarkers of acute and chronic stressor exposure and stress perception. Initial hypotheses in the human literature proposed that higher chronic stress would be associated with sustained elevations in GC and physiological hyperreactivity to stress (hypercortisolism) (Gunnar & Fisher, 2006). However, human studies have found both high and low levels of diurnal cortisol secretion and stress reactivity implicated in healthy but 'at risk' chronic stress samples (Gunnar & Quevedo, 2007; Walker, Pfingst, Carnevali, Sgoifo, & Nalivaiko, 2017) and in physical and psychological disorders (Herane Vives et al., 2015; Kyrou & Tsigos, 2009; Staufenbiel, Penninx, Spijker, Elzinga, & van Rossum, 2013). This has led to multiple theories of chronic stress causing both hypercortisolism as well as hypocortisolism (including diurnal cortisol secretion reductions and hyporeactivity to stress) (Heim, Ehlert, & Hellhammer, 2000). For instance, hypocortisolism has been suggested to be a compensatory down–regulation to facilitate recovery and reduce damage from excessive metabolic expenditure (Del Giudice, Ellis, & Shirtcliff, 2011; Levine, 2006; McEwen & Gianaros, 2011). Therefore, in humans, cortisol secretion has been a less straightforward biomarker of stress than initially hoped. There are presumably several reasons for this, some of which will be discussed below, in particular methodological limitations.

2.6 SES and stress associations with children's cortisol secretion

Several studies have found lower SES or associated chronic stressors to be related to children's basal cortisol secretion, painting a mixed picture of cortisol secretion that is likely, to some extent, to derive from methodological limitations. These studies usually collect serial saliva samples at differing times of the day and report both higher basal cortisol levels (Blair, Granger, et al., 2011; Chen, Cohen, & Miller, 2010; Lupien, King, Meaney, & McEwen, 2000; Lupien et al., 2001) and lower basal cortisol

levels (Badanes, Watamura, & Hankin, 2011; Chen & Paterson, 2006; Kliewer, Reid–Quinones, Shields, & Foutz, 2008; Pagliaccio et al., 2014; Wagner et al., 2016) in lower SES (Badanes et al., 2011; Blair, Granger, et al., 2011; Chen et al., 2010; Chen & Paterson, 2006; Kliewer et al., 2008; Lupien et al., 2000) or more stressed healthy children (Pagliaccio et al., 2014; Wagner et al., 2016). The issue is that basal cortisol measures show considerably lower intra–individual stability (e.g., the intraclass correlation coefficient for a single lunchtime sample is 0.37) than multiple measures of diurnal cortisol collected across several days (Rotenberg, McGrath, Roy–Gagnon, & Tu, 2012; Schmidt–Reinwald et al., 1999), because they are substantially affected by a multitude of factors. For instance, cortisol secretion has a non–linear diurnal secretion pattern, thus, if diurnal secretion patterns have a different shape in low SES, this can lead to the measurement of higher or lower basal levels dependent on the time of day. Confounding variables that influence cortisol levels, such as food intake or acute experiences of stress, also affect basal cortisol levels more than multiple response measures do. Recent recommendations have been made to improve collection and to control for confounds (Smyth, Hucklebridge, Thorn, Evans, & Clow, 2013; see Stalder et al., 2015 for an expert consensus paper on measuring the CAR). More so, cortisol stress reactivity in response to the Trier Social Tress Test for Children (TSST–C; Buske–Kirschbaum et al., 1997) is rarely studied, although its validity compared to other stress challenges and health outcomes has made it the gold standard paradigm in human stress research (Hellhammer, 2011). Nevertheless, these basal cortisol secretion studies provided initial evidence for the notion that SES–related stressors are associated with children's diurnal cortisol secretion in some way. Similar to research on SES–related disparities in cognitive development, the large majority of these studies are genetically uninformed. Behavior genetic twin studies suggest moderate heritability estimates of salivary cortisol (30–40%), but these studies have, again, been limited by failures to account for diurnal cortisol rhythm and small sample sizes (Bartels, de Geus, Kirschbaum, Sluyter, & Boomsma, 2003; Bartels, Van den Berg, Sluyter, Boomsma, & de Geus, 2003; Schreiber et al., 2006; Van Hulle, Shirtcliff, Lemery–Chalfant, & Goldsmith, 2012).

A few studies have looked at dynamic diurnal cortisol secretion or cortisol reactivity to stress as more reliable and valid indices of HPA axis activity than basal cortisol levels in children. Yet, a superfluous amount of statistical indices in the developmental and adult literature have been created to characterize dynamic cortisol secretion (Khoury et al., 2015). From 15 indices commonly used in the literature, a recent cross–validation principal component analysis extracted two dimensions representing 'total cortisol level' and 'change in cortisol', which some of these indices conflate, thereby potentially confounding results if diverging associations are present for level and change (Khoury et al., 2015). Thus, statistical methods must separate differences in total between–person cortisol levels from between–person differences in within–person change over time. Latent–change SEMs provide powerful tools to simultaneously represent between–person differences in cortisol level, change and their covariance, whilst having several other advantages over calculating multiple

difference scores (Hamaker et al., 2015; McArdle, 2009). For example, the number of cortisol outcome variables and thereby the number of statistical comparisons is significantly reduced, because reactivity and recovery measured in multiple cortisol levels can be modeled in one latent slope variable. Second, the characterization of cortisol is informed by the data, because model selection depends on fit to the data. Third, measurement error is explicitly modeled and can, for instance, account for shared residual variance in cortisol samples collected on the same day in diurnal cortisol collections. Lastly, the regression of the SES predictor onto cortisol and the regression of cortisol onto cognition can be represented in one model.

To date, studies report dysregulated diurnal secretory patterns including lower diurnal cortisol slopes (Fisher & Stoolmiller, 2008; Martin, Kim, Bruce, & Fisher, 2014; Martin, Kim, & Fisher, 2016; Wolf et al., 2008) and potentially higher evening levels (Wolf et al., 2008) in association with SES–related stress. We know of no study exploring prepubescent children's CAR along gradients of SES, although one study reports a blunted CAR in early puberty following early life stress (King et al., 2017) and another reports a blunted CAR in low–SES adolescent girls (McFarland & Hayward, 2014). Furthermore, two studies report lower reactivity to stress associated with lower SES (Badanes et al., 2011; Kraft & Luecken, 2009), however null results have also been reported (Hostinar et al., 2015b; Hostinar, Johnson, & Gunnar, 2015a). Lastly, cortisol secretion is strikingly variable between individuals and not all children exposed to stress show HPA axis alterations, which suggests moderation effects (Del Giudice et al., 2011; LaPrairie et al., 2010). In conclusion, it is currently not established whether and how SES–related stress exposure is associated with diurnal cortisol secretion and stress reactivity in children.

2.7 Emerging research on relationships among SES, stress exposure, cortisol secretion, and cognition

Developmental research on the interplay of SES, stress exposure, cortisol secretion, and cognition is beginning to emerge. Childhood cognition is an aspect of human behavior that is closely linked with individual differences in multiple physiological domains, health, and human capital across the lifespan (Briley & Tucker–Drob, 2017; Deary, 2008; Deary, Weiss, & Batty, 2010; Koenen et al., 2009). It can therefore be conceptualized as a moderator or the outcome of SES–related effects.

First, children's cognitive control skills could moderate the association of stress exposure and cortisol secretion. Previous research has shown that behavioral self–regulation is correlated with cortisol secretion at daycare in early childhood and this association is partially accounted for by cognitive control (Blair, Granger, & Razza, 2005; Dettling, Gunnar, & Donzella, 1999; Dettling, Parker, Lane, Sebanc, & Gunnar, 2000; Gunnar, Tout, de Haan, Pierce, & Stansbury, 1997). Cognitive control, which can be defined here as top–down control of goal–directed action (Gross & Thompson, 2007; Ochsner & Gross, 2005) relies heavily on the PFC, which is also involved in

regulating the HPA axis (Lupien et al., 2009). Developmentally, cognitive control shows marked improvement in middle childhood (Davidson et al., 2006; Wright & Diamond, 2014) that is reflected in the development of networks of fronto–parietal and cingulo–opercular brain regions (Dosenbach et al., 2008). Thus, developing cognitive control skills may emerge as a modifier of the effects of stress on HPA axis activity in middle childhood.

Second, animal studies, predominantly in rodents, have documented causal pathways from both chronic stress and GC administration to brain structure and cognitive function, focused heavily on the memory domain (McEwen, 2012). Structurally, chronic stress and GCs lead to neural atrophy in the hippocampus and PFC (McEwen, 2012). Functionally, rodent models and studies of adult humans suggest that GC and memory have an inverted U–shaped relationship (Ursache & Noble, 2016). At both very high levels (de Kloet, Oitzl, & Joëls, 1999; Lupien & Lepage, 2001) and very low levels of GCs (Lupien & Lepage, 2001) long–term potentiation is impaired in the hippocampus and synaptic activity in PFC circuitry decreased, whereas long–term potentiation and synaptic activity is facilitated at medium GC levels (Ramos & Arnsten, 2007; Segal, Richter–Levin, & Maggio, 2010). In more detail, GC action on neural structure and function is shown to play a susceptibility role in a cascade that involves excitatory amino acids, neurotrophins, adhesion molecules, altered GR expression, and neuromodulators such as serotonin (McEwen, 2000). However, rodents and humans differ greatly; for example, humans have a different circadian rhythm, lower levels of GR in the hippocampus but higher levels in the PFC (Sánchez et al., 2001), a substantially larger PFC, and more complex higher–order cognition (Uylings & van Eden, 1990). Therefore, it is not self–evident that this causal pathway found in rodents reproduces in humans. Nevertheless, animal models imply that hippocampal structure and function are critical candidates in linking stress–related SES disparities to individual differences in cognition, especially memory.

Considerably less is known about the relationship between cortisol secretion, neural structure, and cognition in children (Heffelfinger & Newcomer, 2001). Structurally, smaller hippocampal volumes (Brody et al., 2017; Hair et al., 2015; Hanson et al., 2011; Jednoróg et al., 2012; Luby et al., 2013; Noble et al., 2015; Noble, Grieve, et al., 2012; Noble, Houston, et al., 2012; Rao et al., 2010; Yu et al., 2017) and PFC volumes (Hair et al., 2015; Hanson et al., 2012; Noble et al., 2015) found in lower–SES children fit with theories of neural atrophy induced by chronic stress. More so, SES–related volumetric differences in frontal and temporal regions (Hair et al., 2015; Hanson et al., 2012) or in cortical surface area (Noble, Houston, et al., 2015) and thickness (Mackey et al., 2015; Romeo et al., 2017) have been found to be cross–sectionally associated with (Hanson et al., 2012; Mackey et al., 2015; Noble et al., 2015) or longitudinally mediate (Hair et al., 2015) the relationship between SES and executive functions or other cognitive domains and academic achievement. Yet the relation of smaller hippocampal volume to SES–related memory disparities is not established. Although one recent study claims evidence for hippocampal attrition as an SES mediator of memory functioning, the memory task did not actually show SES disparities (Yu et al., 2017). Putative evidence linking

SES dysregulation in cortisol reactivity to hippocampal volume in children has also not been provided.

Furthermore, the literature associating cortisol secretion with cognitive performance is scarce and currently mixed, mirroring research of SES–related stress effects on cortisol secretion. The latter seems to suggest that higher basal cortisol measures (Blair, Granger, et al., 2011; Maldonado et al., 2008; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010; Obradović et al., 2016; Wagner et al., 2016) or both lower and higher basal cortisol levels (Suor, Sturge–Apple, Davies, Cicchetti, & Manning, 2015) are associated with poorer memory, executive function, and intelligence. Another study not looking at SES shows that a lower CAR is associated with poorer prospective memory on that day in children (Bäumler et al., 2014). Further, lower cortisol reactivity to stress has been linked to behavioral and psychiatric problems in children (Kushner, Barrios, Smith, & Dougherty, 2016). Some evidence from intervention research (Bugental, Schwartz, & Lynch, 2010) and longitudinal mediation studies (Blair, Granger, et al., 2011; Suor et al., 2015) suggests children's cortisol secretion is associated with cognition, however these relied on basal cortisol measures. Critically, evidence that SES is associated with dysregulated cortisol secretion, children's hippocampal volume and memory functioning, is absent.

3. Aims of dissertation

As shown in the previous section, the current state of research treats SES indicators as stable predictors, whilst ignoring longitudinal dynamics of change. Moreover, developmental studies of SES–related disparities in cortisol secretion have been limited by a failure to account for the diurnal cortisol rhythm and investigate acute stress reactivity. Research on the association between children's cortisol secretion and cognition, which may both modify embedding of stress and be the outcome of SES–related stress, is in its infancy. It therefore remains to be empirically established whether a stress mechanism may be involved in SES–related cognitive disparities. Therefore, I pursue the following goals in my dissertation:

Aim 1: Explore longitudinal within–person dynamics between income and cognitive development throughout middle childhood and early adolescence (*Paper I*).

Aim 2: Apply advanced statistical methods to reliable and valid measures of dynamic cortisol secretion to investigate whether lower SES and higher stress are associated with dysregulated cortisol secretion in middle childhood (*Paper II & III*).

Aim 3: Test whether cognitive control moderates effects of stress exposure on diurnal cortisol secretion (*Paper II*).

Aim 4: Assess whether SES disparities in children's memory performance are related to differences in cortisol reactivity and hippocampal volume (*Paper III*).

4. Overview of papers

The present dissertation is based on three empirical papers that explore different aspects of the theoretical model depicted below (Figure 1). SES–related disparities in cognition are proposed to derive, in part, from differences in exposure to chronic stress, which affects children's cortisol secretion. Aberrant cortisol secretion is then thought to affect neural function and structure in the hippocampus, thereby influencing cognition, especially memory performance. In addition, cognitive functioning, specifically top–down cognitive control, may also impact how exposure to stress affects the child's response to chronic stress exposure and influences cortisol secretion. First, *Paper I* uncovers longitudinal dynamics between income and cognition in later childhood drawing on data from a large database. Second, *Papers II* and *III* investigate whether higher stress (*II*) and lower SES (*III*) are associated with altered cortisol secretion in two separate middle childhood samples. *Paper II* conceptualizes cognition as a moderator of the embedding of stress exposure, by investigating whether cognitive control moderates effects of stress on diurnal cortisol secretion. *Paper III* further tests the hypothesis that SES disparities in hippocampal–dependent memory are associated with an altered CAR, stress reactivity, and hippocampal volume.

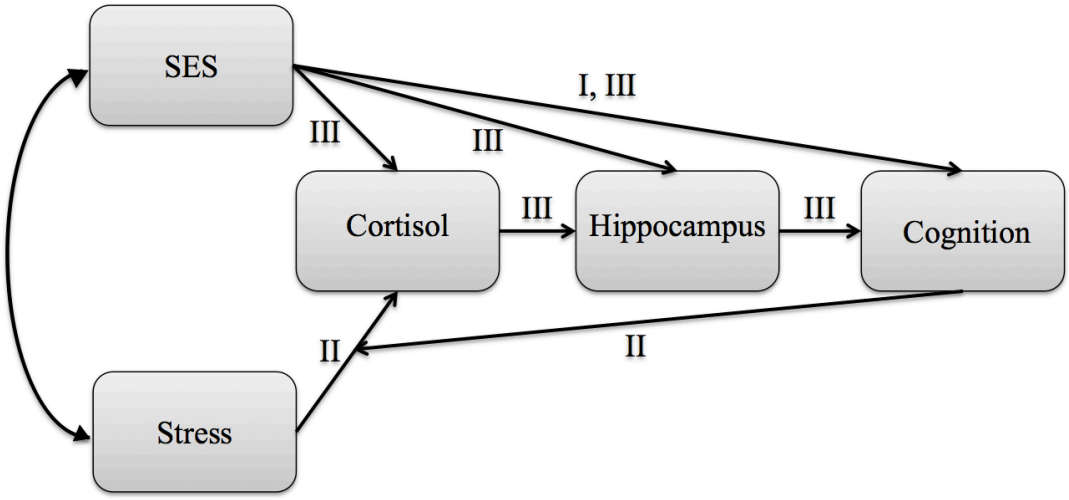

Figure 1. Theoretical dissertation model exploring longitudinal income–cognition dynamics in *Paper I* and stress mechanisms in *Papers II* and *III*. SES = socioeconomic status.

Paper I

Raffington, L., Prindle, J. J., & Shing, Y. L. (2018). Income gains predict cognitive functioning longitudinally throughout later childhood in poor children. *Developmental Psychology*. Advance online publication. http://dx.doi.org/10.1037/dev0000529

Theoretical background

Research has largely ignored longitudinal dynamics of SES indicators and cognitive development, relying instead on between–person comparisons to infer the effects of SES and poverty. Furthermore, correlational studies of between–person differences do not allow causal inferences to be drawn. Very few studies have begun to look at change in income to explore dynamic relationships in non–experimental designs (Duncan et al., 2017, 1998). These suggest that changes in income contribute to cognitive performance in early childhood (Dearing et al., 2001; Mistry et al., 2004). It is less established whether family income continues to predict cognitive growth in later childhood or whether there may even be bidirectional dynamics. Importantly, living in poverty may moderate income–cognition dynamics.

Methods

The data for this study came from the National Institute of Child Health and Human Development Study of Early Child Care and Youth Development, for which participants were recruited at birth from hospitals in several cities across the US in the early 1990's (National Institute of Child Health and Human Development Early Child Care Research Network, 2005). Although the attempt was made to represent the US demographic composition, the sample underrepresents families of lower SES. Nevertheless, 226 (19%) of the 1168 children aged 4 to 12 years contributing data to this study lived below the poverty threshold in at least one wave in the analyzed timeframe. These were defined as 'poor' and compared to children never living below the poverty threshold in any wave in the analyzed timeframe ('never poor'). Two sets of bidirectional dual change score models in seven waves of data evaluated, first, whether one variable's *score* predicted within–person *change* in the other variable from that wave to the next, following modeling convention (Small, Dixon, McArdle, & Grimm, 2013). Second, we explored whether *change* in one variable from one wave to the next predicted the other variable's following *score* to test for effects of income change specifically. Dynamics were explored in children's verbal comprehension and math ability.

Major findings

As previous between–person comparisons have documented, poor children had substantially lower average starting points and cognitive growth slopes through later childhood. The difference in cognition between poor and never poor remained stable over this age range (nearly 1 SD), which is

similar to a study that found parental education and neighborhood quality did not predict rate of developmental change in working memory from age 10 to 13 years (Hackman et al., 2014).

Beyond between–person comparisons, the first set of models showed that within–person income *scores* did not predict cognitive *change*. Remarkably, child cognitive scores positively predicted income change in reverse. This was true for both poor and never poor children's verbal comprehension (standardized $\beta = 0.07$, SE = 0.02, $\Delta\chi^2(1) = 3.93$, $p < 0.05$) and only for never poor children's math ability (standardized $\beta = 0.05$, SE = 0.02, $\Delta\chi^2(1) = 4.05$, $p < 0.05$). We speculate that parents may reduce their work investment, thus reducing income gains, when their children fall behind what their developmental slope would predict. Second, positive within–person income *changes* continued to predict higher cognitive *scores* at the following wave for poor children only (verbal comprehension: standardized $\beta = 0.20$, SE = 0.08, $\Delta\chi^2(1) = 5.92$, $p < 0.05$, math ability: standardized $\beta = 0.12$, SE = 0.06, $\Delta\chi^2(1) = 4\ .54$, $p < 0.05$). This suggests that within–person income gains versus losses continue to predict poor children's cognitive performance in later childhood. This study emphasizes the need to look at the effects of changes in income, to explore bidirectional income–cognition dynamics, and allow for moderation of poverty.

Paper II

Raffington, L., Schmiedek, F., Heim, C., & Shing, Y. L. (2018). Cognitive control moderates parenting stress effects on children's diurnal cortisol. *PLoS ONE, 13*(1): e0191215. http://doi.org/10.1371/journal.pone.0191215

Theoretical background

Parenting stress (Finegood & Blair, 2017; Gershoff et al., 2007; Murgatroyd & Nephew, 2013) and children's self–reported impact of negative life events (LeMoult et al., 2015; Maldonado et al., 2008; Wolf et al., 2008) are two viable indicators of children's stress experiences. Yet, not all children exposed to stress show HPA axis alterations (Del Giudice et al., 2011) and stressors have been associated with higher (Blair, Granger, et al., 2011; Chen et al., 2010; Lupien et al., 2000) and lower basal cortisol levels (Badanes et al., 2011; Chen & Paterson, 2006; Kliewer et al., 2008; Pagliaccio et al., 2014; Wagner et al., 2016). This may be related to issues of reliability in basal cortisol measurements (Rotenberg et al., 2012) or derive from moderation effects (Del Giudice et al., 2011; LaPrairie et al., 2010). Psychological vulnerability and resilience factors are thought to play a vital role in the embedding of stress exposure (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008). Previous research has shown that behavioral self–regulation is correlated with cortisol secretion at daycare in early childhood and is explained in part by cognitive control (Blair et al., 2005; Dettling et al., 1999, 2000; Gunnar et al., 1997). Cognitive control intersects with functioning in multiple behavioral and physiological domains, thereby influencing individual differences in health and human capital across the lifespan (Briley & Tucker–Drob, 2017; Deary, 2008; Deary et al., 2010; Koenen et

al., 2009). Therefore, we hypothesize that cognitive control may act as a moderator of the effects of stress exposure on HPA axis activity. To test this hypothesis, we investigated whether parenting stress and self–reported stress are associated with children's diurnal cortisol secretion and whether this relationship is moderated by cognitive control.

Methods

Participants were recruited from daycare centers across Berlin as part of a longitudinal study. Because exploring SES effects was not the aim of this study, variation in SES was modest and underrepresented low SES. Salivary cortisol concentrations were assessed at the second wave of data collection only from awakening to evening on two weekend days from 53 6–to–7–year–old children as a more stable indicator of diurnal cortisol secretion (Rotenberg et al., 2012). Children completed a cognitive control task and a self–report stress questionnaire with an experimenter, while parents completed a parenting stress inventory (Abidin, 1990). Hierarchical mixed effects models differentiated overall cortisol levels from dynamic cortisol changes over the day and included stress and cognitive control as continuous variables. In addition, we report effects of parenting stress on diurnal cortisol secretion using more common CAR and diurnal slope indices in linear regression models to aid comparability to previous studies.

Major findings

Results revealed that higher parenting stress in parents had a moderate–to–strong association with overall reduced diurnal cortisol levels in children (unstandardized β = -1.46, SE = 0.50, 95% *CI* = -2.02 – -0.91, $p < 0.05$, Cramer's $\varphi(1) = 0.40$), and this effect was moderated by cognitive control (unstandardized β = 1.72, SE = 0.81, 95% *CI* = 0.63 – 2.37, $p < 0.05$, Cramer's $\varphi(1) = 0.30$). Specifically, simple slope analysis showed that at lower levels of child cognitive control higher parenting stress is associated with lower cortisol levels, whereas higher cognitive control ability dissociates parenting stress and cortisol secretion. Although parenting stress was additionally related to a flatter diurnal slope in regression analyses, this was reduced to a non–significant trend when including the significant effect of cortisol level at awakening as recommended for the CAR (Stalder et al., 2015). Therefore, the conservative interpretation of our data derived from converging these regression results with the lack of time–sensitive effects in the hierarchical model is that parenting stress is moderately associated with total cortisol level starting with lower morning cortisol levels that persist to be lower over the rest of the day, but is not significantly associated with cortisol changes in the CAR or diurnal slope. However, given that the study was underpowered (~64%) and SES variance was limited, CAR and diurnal slope null results should be treated with caution. Since self–reported stress was at floor level, did not correlate with parenting stress, and was not associated with diurnal cortisol secretion, this study provides empirical evidence that 6–to–7–year–olds cannot self–report life stress in a meaningful way. In contrast, parent–reported stress appeared as a valid predictor of

children's diurnal cortisol secretion. Taken together, higher cognitive control skills may buffer the effects of parenting stress on their children's HPA axis activity in middle childhood, which is characterized by diurnal hypocortisolism.

Paper III

Raffington, L., Prindle, J., Keresztes, A., Binder, J., Heim, C. & Shing, Y. L. (2018). Blunted cortisol stress reactivity in low-income children relates to lower memory function. *Psychoneuroendocrinology, 90,* 110–121. http://doi.org/10.1016/j.psyneuen.2018.02.002

Theoretical background

Animal models describe a causal pathway from chronic stress exposure to stress system dysregulation to deficient hippocampal development and memory impairments (McEwen, 2000, 2012; Sánchez et al., 2001). Therefore, a similar pathway has been hypothesized to mediate social inequalities in children's cognitive development (Lupien et al., 2001). However, empirical evidence demonstrating these putative links between multiple systems in children is lacking. Aside from basal cortisol measures with low reliability, dynamic diurnal cortisol measures from multiple days and the response to acute stress is rarely studied in children, but has the potential to provide profound evidence for the notion that low–SES is associated with dysregulated stress system functioning. Yet, the majority of acute stress laboratory paradigms fail to actually elicit a mean cortisol increase in children (Gunnar, Talge, & Herrera, 2009). No study to date has linked SES disparities, cortisol responses, hippocampal structure, and memory performance in children.

Methods

Participants were recruited by sending study invitation letters to 7000 families with first graders residing in low and high SES districts of Berlin. This narrow age range was selected to minimize potential moderation effects of developmental age and puberty. Families were screened for exclusion criteria, such as health disorders, steroid medication, trauma exposures and prematurity in children by telephone. First, we followed Farah's (2017) recommendations on exploring effects of SES in a way that increases comparability across studies by reporting the effects of each SES indicator and their composite score, but prioritizing one indicator in our results. The prioritized indicator was income, because income is the most variable SES indicator that may be more closely associated with different levels of family stress including financial strain (Hustedt et al., 2017; Raver et al., 2015) and has been shown to correlate with children's stress reactivity (Badanes et al., 2011; Kraft & Luecken, 2009; but see Hostinar et al., 2015a, 2015b). Although the sample's income distribution included 17% of families living below the poverty threshold, which mirrors population statistics for Berlin, parental education and occupational status suggested that higher SES was overrepresented. Second, we collected state–of–the–art measures of dynamic cortisol responses from 102 6–to–7–year–old

children: (1) the diurnal CAR on two days using electronic time monitoring to ensure compliance and (2) the gold standard of acute stress exposure, the TSST–C (Buske–Kirschbaum et al., 1997) for cortisol stress reactivity (CSR) that elicited a substantial mean cortisol increase. We applied the most recent expert consensus guidelines on cortisol data collection, preprocessing and statistical modeling using latent–change SEMs (Meredith & Tisak, 1990; Stalder et al., 2015). Importantly, these models separate the effects of between–person differences in cortisol *levels* whilst representing dynamic within–person *change* in the CAR and CSR and reduce the number of statistical comparisons. Third, we selected an associative memory task that is known to rely heavily on hippocampal functioning (Kessels, Hobbel, & Postma, 2007; Sander, Werkle–Bergner, Gerjets, Shing, & Lindenberger, 2012) and acquired hippocampal volumes with magnetic resonance imaging on a subsample of these children (n = 60). Statistically, we tested for main effects between SES, cortisol, and cognition associations as well as SES x cortisol interactions as a way to explore gradient versus threshold SES effects without splitting the sample into groups.

Major findings

In sum, we found that lower income has a small–to–moderate association with hyporeactivity in both CAR (standardized $\beta = 0.30$, SE = 0.12, $\Delta\chi^2(1) = 6.12$, $p < 0.05$, Cramer's $\varphi(1) = 0.24$) and CSR (standardized $\beta = 0.25$, SE = 0.09, $\Delta\chi^2(1) = 7.01$, $p < 0.05$, Cramer's $\varphi(1) = 0.26$) as well as with smaller hippocampal volumes (standardized $\beta = 0.22$, SE = 0.10, $\Delta\chi^2(1) = 4.17$, $p < 0.05$, Cramer's $\varphi(1) = 0.26$) and memory (standardized $\beta = 0.44$, SE = 0.18, $\Delta\chi^2(1) = 6.04$, $p < 0.05$, Cramer's $\varphi(1) = 0.24$). There were no main effects of CAR or CSR on memory. While it is still possible that cortisol mediates SES–cognition associations through suppression effects, exploring statistical mediation in this cross–sectional design would be biased, if not misleading (Cole & Maxwell, 2003; Lindenberger, von Oertzen, Ghisletta, & Hertzog, 2011; Maxwell & Cole, 2007; Wu & Zumbo, 2008). Furthermore, there was a significant income x CSR interaction (standardized $\beta = -0.28$, SE = 0.09, $\Delta\chi^2(1) = 9.62$, $p < 0.05$, Cramer's $\varphi(1) = 0.31$), which suggested threshold effects. Specifically, in low–income children only, hyporeactivity to acute stress was associated with impaired hippocampal–dependent memory function. However, smaller hippocampal volumes were not associated with poorer memory performance (standardized $\beta = -0.24$, SE = 0.13, $\Delta\chi^2(1) = 3.15$, $p = 0.07$, Cramer's $\varphi(1) = 0.23$). This suggests that compromised hippocampal function, rather than volume, may underlie lower memory performance, a postulation that awaits empirical scrutiny. Interestingly, the main effect indicating hyporeactivity in both CAR and CSR was specific to using income as the SES predictor. All other effects, including the SES x CSR interaction, were replicated with education and the SES composite score, although occupational status on its own showed no associations with any variables of interest. Several potential covariates of low SES, such as parental smoking, current and past parental psychiatric disorders, birth weight, sleep quality, BMI, ethnicity, as well as performance and mood during the stress task, were statistically ruled out as driving these effects. We thereby delineated a

stress–related mechanism of SES disparities in children's associative memory performance that implies a unique role of cortisol reactivity to acute stress.

5. Discussion

In the following, I will summarize and evaluate the major findings of this dissertation and describe how the presented studies contribute to the existing literature. I present a hypothesized model of cortisol secretion dysregulation in low SES prepubescent children. Then I discuss limitations of the presented studies and identify future research directions, which includes conceptual models of cortisol dysregulation as a function of environmental–and genetically–mediated effects. Finally, I provide general implications of the presented research.

5.1 Summary and evaluation of major findings

Aim 1: Income changes continue to predict cognitive functioning in poor children through later childhood.

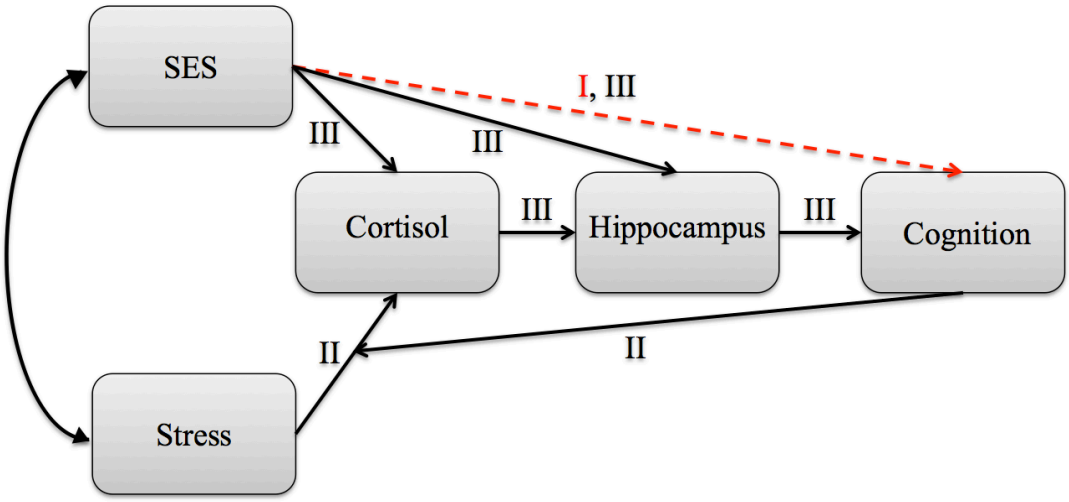

Figure 2. Theoretical dissertation model exploring longitudinal income–cognition dynamics in *Paper I*.

Paper I shows that within–person income gains and losses continue to predict both verbal comprehension and math ability throughout middle childhood and early adolescence for children experiencing poverty in this timespan, but not for children never experiencing poverty during this period. This extends previous research that treated SES indicators as static predictors of child outcomes, inferring the longitudinal effects of income from between–person comparisons (Crosnoe et al., 2010; Lawson & Farah, 2017; von Stumm & Plomin, 2015; Wang et al., 2017). It shows that the effect of income changes predicting child cognition in early childhood (Dearing et al., 2001; Mistry et al., 2004) continues into later childhood and early adolescence for poor children. Our findings highlight the importance of exploring income dynamics in later childhood, because income *changes* predicted cognitive performance for poor children. Since this was consistent for verbal comprehension

and math ability, we assume general cognitive performance of poor children in later childhood is positively predicted by income gains and negatively predicted by income losses. Correspondingly, disparities in different cognitive and academic outcomes along gradients of parental education are largely driven by a single developmental pathway manifest in global cognitive development, yet, math ability has an additional unique pathway (Tucker–Drob, 2013). Although these income changes in later childhood are unlikely to reorder between–person differences (Tucker–Drob & Briley, 2014) and poor children continued to have cognitive growth trajectories substantially lower than never poor children, our finding suggests that within–person cognition is still predicted by within–person income fluctuations for poor families.

We speculate that a multitude of proximal factors could mediate higher cognitive performance in poor children following family income gains or lower cognitive growth following losses, since income does not impinge directly on cognition. Previous studies have provided evidence that family–level environmental factors, such as material goods, parent stress, parent investment, and positive parenting behavior, are cross–sectionally associated with both SES and cognition (Gershoff et al., 2007; Mistry et al., 2004), or longitudinally mediate SES disparities in child executive function in part (Hackman et al., 2015). Importantly, *Paper I* suggests that income gains versus losses matter more to poor children's cognitive performance in later childhood than the previous income level alone. Indeed, income variability in low–income samples is a potent stressor for parents (Hustedt et al., 2017). Furthermore, cognitive stimulation in the home environment varies with changes in family income, particularly in low–income households (Votruba–Drzal, 2003). Similarly, changes in parental behavior mediate the positive effects of moving out of poverty on behavioral disorders (Costello et al., 2003). Following income increases, poor parents may be able to purchase better educational materials at home, better–quality activities, and more nutritious foods (Duncan et al., 2017). The family may also experience a reduction in stress by feeling some relief from financial strain and perhaps moving to a safer neighborhood with positive consequences for children's development (Morrison Gutman, McLoyd, & Tokoyawa, 2005). Alternatively, shared genetic profiles could influence a child's cognitive development and parents' earning potential (see section 5.2).

In contrast, income had no effect on cognitive development in children who in later childhood were never poor, which mirrors results on this sample in early childhood (Dearing et al., 2001). Although cognitive functioning and neural structure are associated with SES across the whole SES continuum in a gradient manner, the gradient seems to be steeper at the lower end (Hair, Hanson, Wolfe, & Pollak, 2015; Noble et al., 2015). This study provides further evidence for distinguishing interpretations based on the analysis of poverty compared to broad SES ranges. Correspondingly, longitudinally measured chronic poverty and financial strain uniquely predict children's executive function development (Raver, Blair, & Willoughby, 2013). We further provide evidence for threshold effects of income–cognition dynamics. However, it is important to note that even in this large sample, SES was negatively skewed, which underestimates effects of income (see section 5.2). Future

longitudinal studies would benefit from collecting multiple SES indicators and proximal mediators at each wave, especially in samples with large between–person and within–person variance in SES (e.g., due to mothers completion of schooling and further educational training during the observation period).

This is the first study to also explore bidirectional dynamics testing for reverse within–person effects of child cognition on parental income. Interestingly, we found that when children's cognitive performance was lower than their developmental trajectory would predict, their parents made less income gains or made losses from that wave to the next. The proximal mechanism that may underlie this effect remains to be elucidated. However, based on the existing literature, we speculate that a plausible mechanism is that children with lower cognitive performance may draw more investments from parents, potentially also affecting their psychological well–being (Miner & Clarke–Stewart, 2008), which in turn lowers income increases (McLoyd, 1990). Findings suggest that parents' feeling that their children are doing well is a strong indicator of self–reported work–family balance (Milkie, Kendig, Nomaguchi, & Denny, 2010) and especially mothers continue to reduce paid work to meet child rearing demands (Bianchi, 2011). Although this reverse effect of cognition on income was present for poor and never poor children's verbal comprehension, it was only significant for never poor children's math ability. Therefore, it may be a stronger effect in more affluent families, who are better able to adjust their work investment depending on their children's needs (Lareau & Weininger, 2008). Deficits in verbal comprehension may be more noticeable to parents than math ability. Thus, lower child cognition may lead parents, especially mothers, to increase their investment in children at the cost of their career investment, thereby reducing family income gains. More generally, these bidirectional dynamics highlight that children are not merely the product of their environment and there are evocative and transactional mechanisms at play in family dynamics that link SES and child development.

Aim 2: Higher parenting stress and lower income is associated with patterns of hypocortisolism in middle childhood.

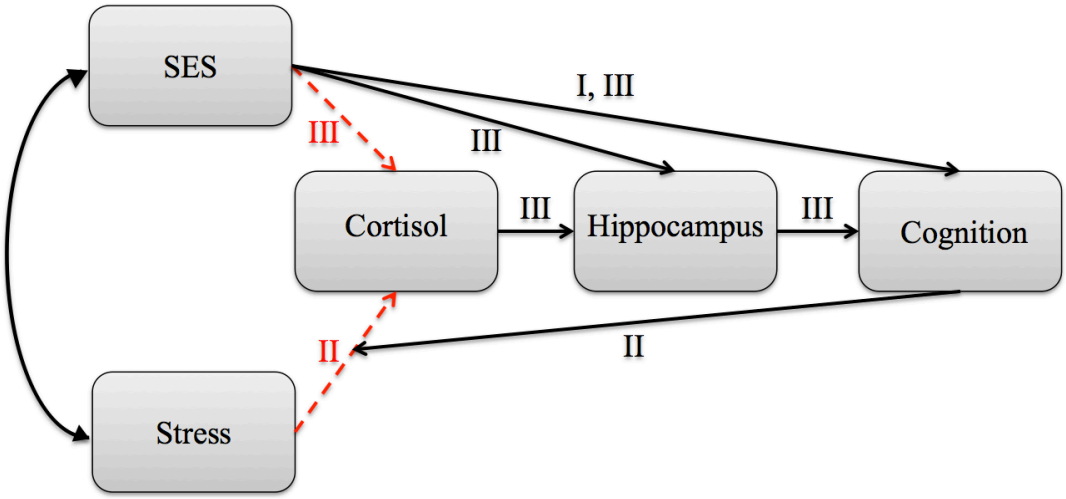

Figure 3. Theoretical dissertation model exploring stress mechanisms in *Papers II* and *III*.

In *Papers II* and *III*, this dissertation reports converging evidence that higher parenting stress and lower income are associated with lower diurnal cortisol levels in middle childhood (*Paper II*) and cortisol hyporeactivity (*Paper III*). Interestingly, the main effect indicating hyporeactivity in both CAR and CSR was specific to using income as the predictor and did not replicate with education, occupation or an SES composite (*Paper III*). Occupational status did not have associations with any variables of interest and is arguably the most difficult SES variable to operationalize, since it has a less straightforward hierarchical structure than education and income. Correspondingly, it is the least used SES indicator in the literature. We hypothesize that this divergence derives from distributional properties of these SES indicators, as education and occupational status overrepresented higher SES. Income is the most variable SES indicator (Duncan, Ziol–Guest, & Kalil, 2010), the only interval variable, and may be more closely associated with different levels of family stress related to financial strain (Hustedt et al., 2017; Raver et al., 2015), even in relatively well–educated parents with socially recognized occupations. Thus, we corroborate previous literature that SES indicators are only moderately correlated among each other in more modestly varying SES samples and have diverging effects on child outcomes (Farah et al., 2008; Noble et al., 2015). Our results based on reliable and valid cortisol measurements significantly contributes to the mixed literature showing higher and lower basal cortisol patterns in association with low SES or related stressors (Badanes et al., 2011; Blair, Granger, et al., 2011; Chen et al., 2010; Chen & Paterson, 2006; Kliewer et al., 2008; Lupien et al., 2000; Wagner et al., 2016).

Furthermore, *Paper III* reports a trend–level negative association of income with pre–stress levels that could be indicative of higher basal afternoon cortisol levels in low–income children, potentially deriving from a flatter diurnal cortisol slope previously reported (Fisher & Stoolmiller,

2008; Martin et al., 2014, 2016; Wolf et al., 2008). However, a number of other factors, such as anticipatory reactions and previous acute stress experiences, which are more frequent in lower SES, could also cause basal increases. Correspondingly, pre–stress levels were not correlated with CSR. This exemplifies that measuring reliable and valid dynamic cortisol levels, for instance on multiple days or in response to acute stress, are essential for between–person comparisons. Additionally, statistical indices that conflate level and change would have missed income effects, given diverging associations with intercept and slope. In this dissertation I combined such measures of cortisol secretion and used advanced statistical tools that separate between–person levels from dynamic within–person change, which suggests that low income–related stress is associated with cortisol secretion attenuation.

More specifically, based on the findings of this dissertation and other recent publications, I conclude that in middle childhood, lower income and higher stress are predominantly associated with patterns of hypocortisolism in short–term diurnal salivary profiles (e.g. 2 days) and stress reactivity. As illustrated in Figure 4, I propose that prepubescent children's cortisol secretion in lower SES is profiled by a hyporeactive CAR slope (*Paper III*) (McFarland & Hayward, 2014), flatter diurnal slopes that could appear as higher afternoon basal cortisol levels (Fisher & Stoolmiller, 2008; Martin et al., 2014, 2016; Wolf et al., 2008; but see Hustedt et al., 2017), and lower total diurnal cortisol levels (*Paper II*). A flatter diurnal slope may also contain elements of hypercortisolism if evidence of higher bedtime cortisol levels is replicated (Wolf et al., 2008). Furthermore, cortisol responses to acute psychosocial stress (TSST–C) are hyporeactive (*Paper III*) (Badanes et al., 2011; Kraft & Luecken, 2009; but see Hostinar et al., 2015a, 2015b). Correspondingly, in adults chronically low SES from infancy through early adulthood predicts the lowest CARs, flattest diurnal slopes, highest bedtime cortisol levels and lowest total cortisol levels across the day (Desantis, Kuzawa, & Adam, 2015). We speculate that *Paper II* did not replicate significant time–sensitive slope differences over the day, although there was some evidence for a flatter diurnal slope, because of limited SES variance and a small sample size. While total cortisol level reaches moderate stability with only 2 days of measurement, the CAR and diurnal slope need more than 3 days, and bedtime levels need 4 days (Ross, Murphy, Adam, Chen, & Miller, 2014; Rotenberg et al., 2012). Thus, null results in the CAR and diurnal slope should be treated with caution (*Paper II*). Furthermore, a recent study in young adults suggests that higher rumination is associated with blunted reactivity to the typical stressful TSST, but higher cortisol reactivity to an intermediately difficult low–stress TSST (Vrshek-Schallhorn, Avery, Ditcheva, & Sapuram, 2018). Thus, low–SES children may similarly show blunted reactivity to acute stress, as suggested by our TSST–C study (*Paper III*), but heightened reactivity to low–stress challenging situations. This may explain diverging findings where lower income is associated with lower executive functions only in children with *increased* reactivity to challenging tasks that are not stressful enough to elicit a mean group cortisol increase (Obradović et al., 2016).

Additionally, emerging research on long–term cumulative cortisol levels measured with high reliability in hair (across 2–3 months) suggest *heightened* levels in low SES children (Flom, St. John, Meyer, & Tarullo, 2017; Rippe et al., 2016; Tucker–Drob et al., 2017; Vaghri et al., 2013). In contrast to enthusiastic interpretations of hair cortisol being a biomarker of cumulative chronic stress exposure, a recent study showed that genetic factors account for approximately half of the variation in cortisol, but lower SES was also associated with steeper increases in cortisol with age cross–sectionally (Tucker–Drob et al., 2017). Therefore, more frequent low–stress and acute stress challenges and higher nocturnal cortisol secretion as a continuation of higher evening levels, and shared influence of stress–system genes could associate higher long–term cumulative cortisol levels with predominantly hyporeactive diurnal and acute stress cortisol secretion. We know of no study exploring hair and salivary associations, CSR frequency or nocturnal cortisol secretion in low–SES children.

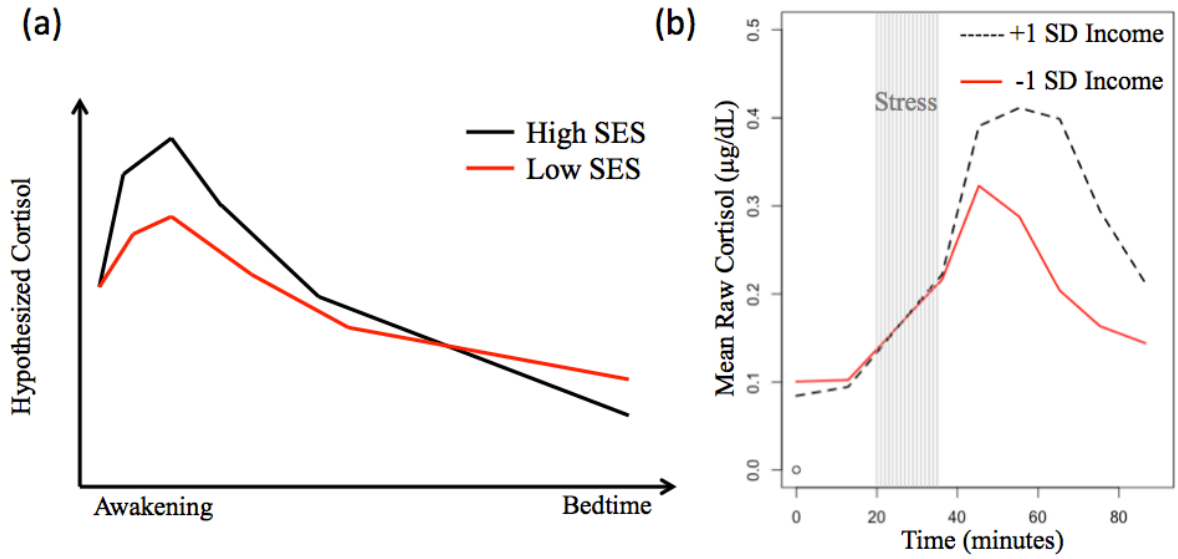

Figure 4. Hypothesized profile (a) of low SES diurnal cortisol secretion marked by a blunted CAR slope, flatter diurnal slope, higher evening levels, and reduced total diurnal level. In addition, responses to acute stress are hyporeactive (b, actual data from *Paper III*). Lastly, cumulative cortisol levels measurable in hair may be increased (not shown).

We speculate that hypocortisolism in short–term diurnal salivary cortisol measures reflects lower steady–state HPA axis activity at the level of the pituitary in response to chronic activation from the hypothalamus (Fries, Hesse, Hellhammer, & Hellhammer, 2005). Hyporeactivity could be a distinct allostatic adaptation of the HPA axis to higher chronic stress exposure to protect from excessive expenditure of metabolic resources in the face of recurrent stress and to reduce the risk of neural damage from overexposure to cortisol (Fries, Dettenborn, & Kirschbaum, 2009; Gunnar & Quevedo, 2007; Levine, 2006). It remains to be established whether cumulative chronic stress and heightened cumulative cortisol levels (Flom et al., 2017; Rippe et al., 2016; Tucker–Drob et al., 2017;

Vaghri et al., 2013) co–occur or even precede patterns of hypocortisolism in low–SES children. An environmental chronic stress mechanism has garnered some support in longitudinally mediating higher and lower basal cortisol levels (Blair, Raver, Granger, Mills–Koonce, & Hibel, 2011; Suor et al., 2015). Importantly, family risk and experiential canalization models propose that specific aspects of the *early* caregiving environment are operative in shaping HPA axis activity in a direction that maximizes functioning within a specific expected environment (Blair & Raver, 2012). Thus, the reported pattern of cortisol dysregulation in middle childhood may be preceded by higher chronic stress in early childhood (see section 5.2 for further discussion of early childhood effects). More generally, SES–related patterns of hyporeactive cortisol secretion may present a functional adaptation to more stressful environments. Additionally, genetic effects may substantially explain variation in cortisol secretion (Bartels, de Geus, et al., 2003; Bartels, Van den Berg, et al., 2003; Schreiber et al., 2006; Tucker-Drob et al., 2017; Van Hulle et al., 2012), although genetically informed research on dynamic cortisol reactivity is lacking.

The hypothesized profile of lower–SES pre–pubescent children's cortisol secretion dysregulation awaits further empirical scrutiny. In general, the developmental cortisol literature has been characterized by unreliable cortisol measurements, underpowered samples, multiple statistical comparisons, age variability, and high researcher variability in what aspect of the cortisol response is modeled and what SES predictor is utilized. Moving forward, it is imperative that studies adequately sample from low SES strata, report the replicability of their results with different SES indicators, control for age and puberty, collect reliable diurnal cortisol measures from multiple days (minimum 2, but preferably 4 or more to explore reliable evening levels; Rotenberg, McGrath, Roy–Gagnon, & Tu, 2012) using data quality controls (e.g., timing compliance monitoring), explore acute stress reactivity and habituation (preferably with the TSST–C), and statistically disentangle cortisol level and change parameters. Additional expert consensus guidelines on the study of diurnal slope, CSR, and hair cortisol would decrease researcher variability and multiple comparisons, as has been done for the CAR (Stalder et al., 2015).

Aim 3: Cognitive control moderates effects of stress exposure on total diurnal cortisol levels.

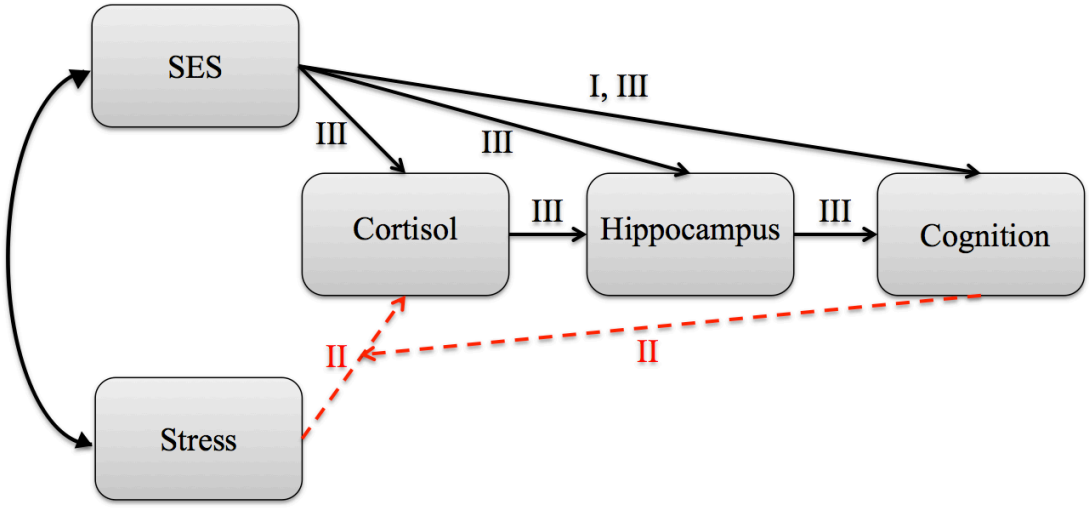

Figure 5. Theoretical dissertation model exploring stress mechanisms in *Paper II*.

In *Paper II,* we found that cognitive control modified the relationship between parenting stress and diurnal cortisol. Specifically, at lower levels of child cognitive control higher parenting stress is associated with lower cortisol levels, whereas at higher cognitive control skills parenting stress and children's cortisol secretion were not reliably associated. Presumably, higher cognitive control skills are related to higher emotional self–regulation (Blair et al., 2005), which enables these children to ward off stress responses otherwise transferred by their parents. Similarly, high effortful control, low negative affect, and low surgency mitigated the negative associations between SES risks and both reading and math development in a very large sample (Wang et al., 2016). These finding suggests that executive functions also need to be considered as moderators, not just outcomes, of SES–related stress effects on HPA axis activity and cognition. Even more so, if cognitive control is both a moderator and outcome of chronic stress exposure, this could indicate a snowball effect of psychological vulnerability leading to lower cognition, such that lower cognitive control facilitates the embedding of chronic stress, leading to lower cognitive control and so on. A longitudinal DCSM could test for such bidirectional dynamics. Since HPA axis activity is also associated with psychiatric and health disorders (LeMoult et al., 2015), these results may be informative in non–cognitive domains. Therefore, investigating psychological vulnerability and resilience factors in relation to stress is an important area of ongoing research (Heim et al., 2008). A central challenge will be to relate such cognitive moderators to genetic effects, such as those hypothesized to make some individuals more susceptible to their environments.

Aim 4: Blunted cortisol reactivity to stress is uniquely associated with hippocampal–dependent memory performance in low–income children, who have smaller hippocampal volumes.

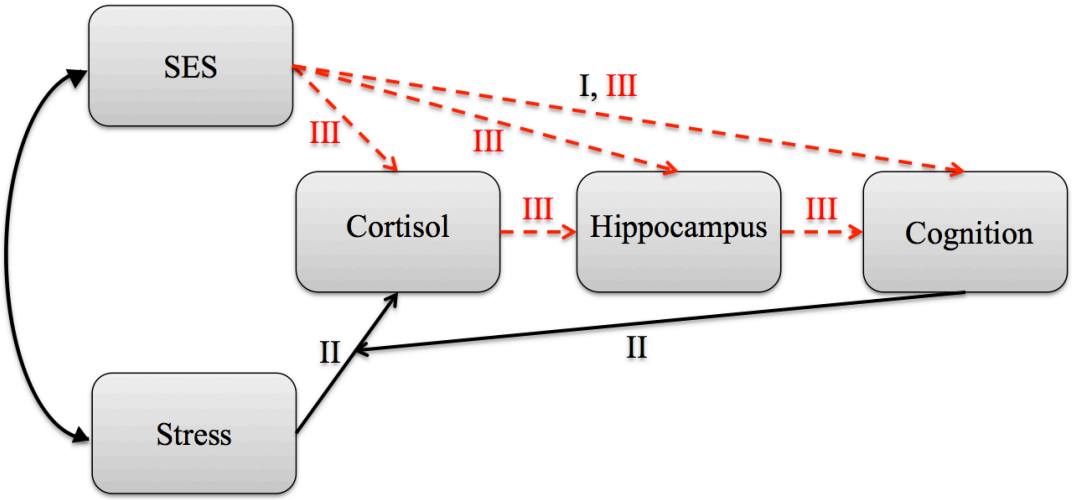

Figure 6. Theoretical dissertation model exploring stress mechanisms in *Paper III*.

Furthermore, in *Paper III* there was a significant income x CSR interaction, which replicated with other SES predictors. Specifically, hyporeactivity in CSR was related to lower associative memory among lower–income children, whereas there was no reliable association of CSR and memory among higher–income children. This provides converging evidence with *Paper I* that there are threshold effects in the association of SES and cognition in middle childhood, suggesting that cortisol dysregulation may only be associated with memory at the lower end of the SES spectrum. In higher SES families, lower stress exposure may partly dissociate cortisol–cognition associations even though main effects of SES on cognition are still found (see section 5.2 for further discussion on threshold effects).

Additionally, hyporeactivity in low–income children's CAR was not related to memory. Since the CAR and CSR were only moderately correlated and only the CSR showed sex differences, we believe the CAR and CSR are largely distinct indices of HPA axis activity (Bouma, Riese, Ormel, Verhulst, & Oldehinkel, 2009; Schmidt–Reinwald et al., 1999) that are differentially associated with different forms of memory. The relationship between associative memory and cortisol may be specific to CSR, because CSR is a response to experiencing a lack of control and social–evaluative threat in response to a cognitively challenging task (Buske–Kirschbaum et al., 1997; Smyth et al., 2013), whereas the CAR is not. Correspondingly, the medial PFC seems to be involved only in stress–induced HPA axis activity, but not in the regulation of diurnal HPA axis activity (Herman, Ostrander, Mueller, & Figueiredo, 2005). Hence, associative memory functioning and CSR may show more neural correlates, whereas the CAR seems more closely associated with preparing the brain for upcoming challenges, including remembering what actions must be performed that day (Bäumler et al., 2014; Clow et al., 2010). Alternatively, CSR is just a more valid measure of HPA axis functioning.

Consequently, our results suggest a stress–hyporeactive cortisol mechanism of income disparities in low–income children's associative memory performance. Notably, evidence of a stress mechanism does not warrant environmentally– or genetically–mediated assumptions of causality (see 5.2).

The association of CSR hyporeactivity with low–income children's associative memory may reflect impairment in long–term potentiation in the hippocampus and a decrease in synaptic activity in PFC circuitry associated with low levels of GCs (Lupien & Lepage, 2001; Ramos & Arnsten, 2007; Segal et al., 2010). Partially confirming our third hypothesis, lower income (as well as the education and SES composite score) was associated with smaller hippocampal volume as has been found in previous studies (Brody et al., 2017; Hair et al., 2015; Hanson et al., 2011; Jednoróg et al., 2012; Luby et al., 2013; Noble et al., 2015; Noble, Grieve, et al., 2012; Noble, Houston, et al., 2012; Rao et al., 2010; Yu et al., 2017). However, hippocampal volume had a trend–level *negative* association with memory, which deserves some consideration, in view of a meta–analysis reporting negative associations between hippocampal volume and memory in children and young adults (Van Petten, 2004). Thus, these smaller volumes are unlikely to link to lower memory in lower–income children. In contrast, SES–related volumetric differences in frontal and temporal regions (Hair et al., 2015; Hanson et al., 2012) or in cortical surface area (Noble et al., 2015) and thickness (Mackey et al., 2015; Romeo et al., 2017) have been found to be cross–sectionally associated with (Hanson et al., 2012; Mackey et al., 2015; Noble et al., 2015) or longitudinally mediate (Hair et al., 2015) the relationship between SES and executive functions or other cognitive domains and academic achievement. Smaller hippocampal volume could simply derive from smaller global gray matter volume seen in lower–SES children (Betancourt et al., 2016; Hair et al., 2015; Hanson et al., 2012; Noble et al., 2015), instead of providing evidence for a chronic stress mechanism located in the hippocampus.

Research on environmental SES mediators and hippocampal volume could shed more light on this issue: One longitudinal study suggests life stress and parenting quality together fully mediate SES effects on hippocampal volume whilst controlling for gray matter volume, although this study oversamples preschoolers with depression (Luby et al., 2013). Similarly, a parenting intervention buffered against the effects of poverty on left hippocampus subfield reductions observed in the control group, though regional specificity was not established (Brody et al., 2017). In contrast, a third longitudinal study suggests that *lower* parental nurturance is associated with *larger* hippocampal volume and there is no effect of environmental stimulation measures (Rao et al., 2010). Thus, SES–related environmental stress may be associated with hippocampal volume, but current evidence is inconsistent. Similar to findings on cognition, the SES–hippocampus correlation could also derive from genetic effects as heritability estimates for hippocampus volume are around 40–69% (Peper, Brouwer, Boomsma, Kahn, & Hulshoff Pol, 2007). Moreover, in *Paper III,* cortisol reactivity dysregulation was not correlated with hippocampal volume, similar to a previous study (Sheridan, How, Araujo, Schamber, & Nelson, 2013). In contrast, higher *early* childhood basal cortisol levels mediate genetic risk (polymorphisms measured in four stress–system genes) and early life stress

effects on smaller hippocampal volumes in later childhood (Pagliaccio et al., 2014). This may indicate that cortisol secretion in early childhood is more important for later neural development. Importantly, without reliable evidence that these SES–related hippocampal differences link to underlying memory deficits in lower SES children, we are relying on reverse inference using between–person neural differences to infer participants' cognitive abilities (Ellwood–Lowe, Sacchet, & Gotlib, 2016; Poldrack, 2011). Thus, while some evidence suggests environmental stress and parenting may mediate SES disparities in hippocampal volume, it is currently not established that smaller hippocampal volume observed in low SES indicates effects of cortisol dysregulation on a stress–sensitive region that mediates memory disparities in children.

Furthermore, emerging evidence suggests that SES and chronic stress may moderate functional brain–behavior relations (Farah, 2017; Vogel & Schwabe, 2016). SES seems to have a selective influence on hippocampal– and PFC–dependent memory, showing less hippocampal activation in associative memory in children (Sheridan et al., 2013), but sparing striatal–dependent procedural memory in adolescents (Leonard, Mackey, Finn, & Gabrieli, 2015). In adults, childhood poverty moderates the engagement of the hippocampus in recognition memory, such that more hippocampal activation is associated with lower recognition memory in previously poor, while the reverse is true for those who were never poor (Duval et al., 2017). The notion that SES moderates hippocampal engagement in memory is strengthened by experimental evidence advocating that stress shifts usage of a hippocampal–based, flexible system to the striatum–based, rigid habit memory system that is thought to have benefits in high stress environments (Schwabe, Bohringer, Chatterjee, & Schachinger, 2008; Schwabe & Wolf, 2011, 2013; Vogel & Schwabe, 2016). Further, stress may alter the expression of plasticity moderators, such as BDNF, which are critically involved in hippocampal plasticity for learning (Gray, Milner, & McEwen, 2013).

Future work is needed to explore functional hippocampal, striatal, and PFC activity and connectivity that may be differentially engaged in low–SES children's associative memory performance due to stress. To better understand this process, physiological stress effects beyond cortisol will need to be explored, since a meta–analysis suggests that acute stress impairs working memory and cognitive flexibility through mechanisms aside from, or in addition to, cortisol to produce a state characterized by more reactive processing of salient stimuli but greater control over actions (Shields, Sazma, & Yonelinas, 2016), such as dopamine and serotonin (LaPrairie et al., 2010). Similar to the study of structural differences, functional brain activation needs to be linked to differences in cognitive performance to make sense of them, but will also need to match children in performance to explore moderation of brain–cognition relationships (Fair, Brown, Petersen, & Schlaggar, 2006). Thus, genetic and environmental differences associated with SES may moderate which neural systems are employed in a cognitive task and to what degree, but without behavioral evidence to suggest so, these differences need not imply a deficit in lower–SES children (Ellwood-Lowe et al., 2016; Kraus, Piff, Mendoza-Denton, Rheinschmidt, & Keltner, 2012).

5.2 Limitations of the reported studies and future research directions

Lack of longitudinal cortisol data spanning early childhood

The lack of multi–wave longitudinal developmental research spanning early childhood examining the influence of SES adversity on cortisol secretion severely limits the feasibility of causal assumptions. For example, in *Paper II*, which is cross–sectional, it is also possible that children with lower diurnal cortisol levels increase their parents' stress, because of, say, associated behavioral problems. Related literature of emotion regulation provides evidence for transactional longitudinal relationships between maternal sensitivity and stress, and child emotion regulation and behavioral problems in early childhood (Finegood, Blair, Granger, Hibel, & Mills–Koonce, 2016; Williford, Calkins, & Keane, 2007). Indeed, children's ANS activity during an emotional frustration task is both predicted by early childhood maternal sensitivity and predicts middle childhood maternal sensitivity (Perry, Mackler, Calkins, & Keane, 2014). Additionally, it is not yet empirically established whether cortisol dysregulation precedes cognitive impairment or cognitive impairment precedes cortisol dysregulation. Accordingly, *Paper II* also suggests that cognitive control moderates the association of stress and diurnal cortisol secretion, which may imply reciprocal cortisol–cognition dynamics that need to be studied in longitudinal designs (Lindenberger et al., 2011). The cross–sectional data of *Paper III* also did not allow us to look at income changes, which *Paper I* suggests is an important predictor of child cognition.

Specifically, the greatest proportions of causal effects of environmental disadvantage are most likely to occur early in ontogeny (Heckman, 2006; Tucker–Drob & Briley, 2014), including prenatal and postnatal childhood 'programming effects' during sensitive periods leading to long–lasting change (Buss et al., 2012; Lupien et al., 2009; Rash et al., 2016). The early environment may shape HPA axis activity in a direction that maximizes functioning within a specific expected environment (Blair & Raver, 2012). HPA axis dysregulation measurable in middle childhood may therefore derive from early childhood stress rather than co–occurring stress. For example, prenatal maternal mood is prospectively and modestly associated with a blunted CAR and flatter diurnal slope in adolescents (O'Donnell et al., 2013) and higher prenatal cortisol exposure measured in amniotic fluid is prospectively associated with higher pre–stress cortisol values and blunted reactivity to separation–reunion stress at 17 months (O'Connor, Bergman, Sarkar, & Glover, 2013). The neural and cognitive effects of stress exposure are also thought to differ depending on when they occur, rendering the brain regions and networks that are rapidly developing at the time of the exposure more sensitive (Lupien et al., 2009). Lower–SES children show a steeper trajectory of normal cortical thinning in early and middle childhood that levels off in adolescence, whereas thinning is more gradual and linear among higher–SES children (Piccolo, Merz, He, Sowell, & Noble, 2016). Furthermore, while the gap in longitudinally measured cognition between higher and lower SES seems to be stable in older children (*Paper III*) (Hackman et al., 2014), it greatly increases in early childhood (von Stumm & Plomin,

2015). Thus, SES–related stress in early childhood may sensitize the HPA axis and initiate maturational neural processes along a different trajectory to maximize functioning in those environments, potentially at the cost of certain cognitive functions preferred in cognitive testing and academic contexts. This type of environmental stress attunement is likely to occur in interaction with genetic influences via epigenetic modifications of multiple genes involved in regulating the stress system and plasticity modulators (Heim & Binder, 2012; Klengel et al., 2013; Meaney, 2010).

Yet, both the HPA axis and developing brain are systems evolved to respond to environmental changes across the life course. Evidence is accumulating that the association of poverty and stress with HPA axis dysregulation is developmentally sensitive. Cross–sectionally, higher early life stress is related to a lower CAR in earlier puberty and a higher CAR in later puberty (King et al., 2017). Longitudinally measured poverty exposure in infancy (0–1 years) and adolescence (11–15 years), but not childhood (1–11 years), is associated with a lower CAR at 15 years among girls (McFarland & Hayward, 2013). More generally, the HPA axis is a system evolved to allow us to tackle the challenges our environments afford throughout life (Danese & McEwen, 2012). Similarly, the PFC does not reach maturity until young adulthood (Huttenlocher, 2009; Lenroot & Giedd, 2006; Thatcher, 1991) and the hippocampus continually adjusts to the environment throughout the lifespan (Eriksson et al., 1998; Freund et al., 2013; Lenroot & Giedd, 2006). Correspondingly, *Paper I* shows income gains and losses continue to predict poor children's cognition in later childhood and early adolescence. Thus, both endocrine and neural development is characterized by phases of heightened environmental sensitivity, especially early in developmental ontogeny, but remain plastic systems. This suggests that environmentally–mediated effects of SES are unlikely to derive solely from differences in prenatal and early postnatal experiences. Because cross–sectional designs inadequately represent developmental change that occurs within individuals over time and can bias statistical mediation, the only appropriate test of such mechanisms is longitudinal in nature (Cole & Maxwell, 2003; Lindenberger et al., 2011; Maxwell & Cole, 2007; Wu & Zumbo, 2008). Notably, repeated measurement of the TSST–C is generally regarded as a measure of stress habituation rather than a repeated–measure, which may make longitudinal TSST–C studies more complex to interpret.

Sampling window

Furthermore, associations of SES, cortisol and child cognition are unlikely to be completely linear across the population. First, evidence suggests SES–related disparities in cognition are strongest in low SES with diminishing returns at higher SES (Hair, Hanson, Wolfe, & Pollak, 2015; Noble et al., 2015) (see Figure 3, left panel). However, studies differ considerably in the SES variance they capture, with poor subjects scarce or nonexistent in many samples. Yet, *Paper I* shows that distinguishing effects of poverty over and above SES gradients is important for income–cognition dynamics. Furthermore, *Paper III* suggests income moderates the cortisol–memory association and that CSR

hyporeactivity is associated with poorer memory in lower–income children only. Similarly, a meta–analysis suggests that the SES variance sampled in individual studies moderates effect sizes between SES and executive functions (Lawson, Hook, et al., 2017).

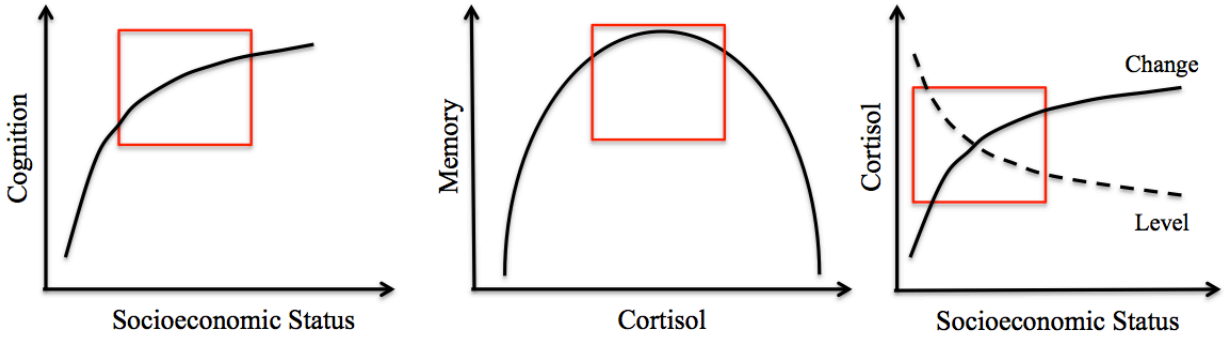

Figure 3. Potential associations of SES, cognition, and cortisol secretion ('change' as short–term dynamic responses in diurnal profiles or stress reactivity and 'level' as cumulative levels over months) that are dependent on the sampling window (red box) of individual studies. The left panel shows SES–related disparities in cognition are strongest in low SES with diminishing returns at higher SES. The mid panel suggests an inverted U–shape relationship between cortisol levels and memory. The right panel proposes lower cortisol change but higher cumulative levels at lower SES, although these associations are largely unknown.

Second, adult studies suggest an inverted U–shape relationship between cortisol and memory (Figure 3, mid panel) (Domes, Rothfischer, Reichwald, & Hautzinger, 2005; Ursache & Noble, 2016). If cortisol secretion is associated with SES–related stress (*Papers II & III*), then the relationship of cortisol levels and cognition will depend on SES variance represented in the study unless cortisol levels are manipulated. Third, the population–wide association of SES and cortisol secretion, especially across developmental time, is largely unknown. SES–related stress effects may be associated with hyporeactive short–term salivary cortisol profiles and higher cumulative hair cortisol levels in middle childhood (Figure 3, right panel). However, even a curvilinear relation between environmental stress (low stress, moderate stress, dangerous environment, traumatic stress) and stress responsivity has been proposed by the Adaptive Calibration Model (Del Giudice et al., 2011; Ellis, Oldehinkel, & Nederhof, 2017). Future research must examine diurnal cortisol secretion, stress reactivity, and hair cortisol with wide SES variance and adequate power to look at developmental age effects. Lastly, cognition and neural structure do not necessarily have a positive linear association (not shown). For example, gray matter volume follows an inverted U–shape trajectory over developmental time (Lenroot & Giedd, 2006) and skill acquisition (Wenger, Brozzoli, Lindenberger, & Lövdén, 2017), whilst cognitive performance is rapidly increasing monotonically (Ferrer & McArdle, 2004). Since SES seems to moderate non–linear structural development (Piccolo et al., 2016), this can severely impact cross–sectional comparisons. More generally, the association of structural plasticity

and higher–order cognition is far from understood (Lindenberger, Wenger, & Lövdén, 2017; Wenger et al., 2017), which poses a further challenge to the neuroscientific study of SES–related disparities in interpreting structural differences. These hypothesized bivariate associations imply that the sampling window of individual studies may critically influence the presence and directionality of effects. Thus, understanding SES–related effects cannot be ignored if we seek to comprehend human development in a generalizable way.

Future studies should make the substantial effort to over–sample at the lowest SES strata to disentangle effects of SES and poverty and resolve threshold versus gradient effects on HPA axis and cognitive functioning. Recruitment of low–SES participants is challenging, in part because they tend to be more isolated from the institutions at which research is conducted and through which participants are most easily recruited (e.g., schools and workplaces; Wilson, 1987). Thus, utilizing research designs that successfully sample wide SES variance and oversampling lower SES is critical. Furthermore, even though SES–related disparities in child outcomes are nearly ubiquitously found, there is no reason to believe the proximal mechanisms are uniform across samples of differing racial and ethnic groups within one country or cross–nationally (Hackman & Farah, 2009). Structural neuroimaging studies need to be especially cautious of cross–sectional comparisons across differing developmental ages and need not limit themselves to gray matter. Lastly, meta–analytic techniques provide a good solution to sampling constraints of individual studies and can account for publication bias and cross–cultural differences.

Omitted variable bias and operationalizing SES effects

The effects ascribed to SES likely reflect the impact of a variety of highly correlated genetic and environmental factors that change over time. *Paper I* looks at cross–lagged coupled change to provide a lower bound estimate of income's effects. However, an omitted variable that is closely coupled to both variables of interest could still account for results. For example, improving maternal mental health could influence both income gains and losses as well as parenting practices that affect children's cognitive performance (McLoyd, 1990). Similarly, cross–sectional *Papers II* and *III* do not support assumptions of causality. However, given that SES indicators are not psychological constructs and necessarily rely on mediator effects, the notion of omitted variables is somewhat misrepresentative of the larger issue of operationalizing SES and its mediators.

There is ongoing debate about how to operationalize SES, because the correlation of SES indicators among each other and with environmental indices is far from perfect (see section 2.3). Depending on what is measured—income, education, or other dimensions of SES—proximal mechanisms may differ, and different interventions may be indicated (Johnson, Riis, & Noble, 2016). Nevertheless, there are variables assumed to be mediators of SES (e.g., stress and enrichment) and variables not studied as part of the SES concept (e.g., maltreatment) – these should be defined and

controlled for a priori or statistically accounted for a posteriori. *Paper II* provides empirical evidence that measuring self–reported stress perception as a more proximal mechanism of SES–related stress exposure is not viable through young (under 8–year–old) children's self–reports, presumably due to a lack of reliability and introspective abilities (Bögels et al., 2001; Caster et al., 1999; Tein et al., 1994; Weissman et al., 1980). Parent–reported stress of themselves or of their children presents a more promising proxy of SES–related stress associations with children's diurnal cortisol secretion (*Paper II*) and cognitive functioning (Gershoff et al., 2007). However, even adults show a discrepancy between their self–reported stress and their *own* CSR (Juliane Hellhammer & Schubert, 2012; Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004). Additionally, parent–reports and experimenter ratings differ in how well they characterize between–person differences according to the sampling window (Votruba‐Drzal, 2003). Thus, more proximal environmental measures, most commonly collected by parental report, are also distal predictors of children's lived daily and cumulative stressor exposure and stress perception. The fact that income predicts HPA axis functioning in *Paper III* suggests that stress mechanisms are at play, but these need not imply environmental stress effects. They could derive from environmentally– or genetically–mediated differences correlated with SES aside from stress (e.g., genes, toxins, nutrition, a predisposition to psychiatric disorders) that could potentially also cause patterns of hypocortisolism.

Thus, it remains largely obscure what may explain SES–cognition associations that combine gradient and threshold effects. Presumably, differently weighted combinations of causes operate at different levels of SES (Farah, 2017), such as cortisol dysregulation only being related to memory in low–income children where chronic stress may be very high (*Paper III*). Similarly, the degree of genetically–mediated effects may also vary along SES strata (Tucker-Drob & Bates, 2016) (see below). Thus, future research should explore moderation effects of SES. However, given that SES is not a 'natural kind' (Quine, 1969), it may prove very difficult, if not impossible, to disentangle environmental and genetic pathways that explain SES–cognition correlations across the entire popoulation. On the other hand, disregarding that children live in vastly different environments with adverse circumstances that tend to cluster together, and relying on samples that are most easily recruited (usually of higher SES) also severely limits our comprehension of human development. Encouragingly, this nascent field enthusiastically engages in critical discussion of its research and limitations (e.g., Ellwood–Lowe, Sacchet, & Gotlib, 2016; Farah, 2017; Hackman & Farah, 2009; Johnson et al., 2016).

Most crucially, the study of SES needs more (quasi–) experimental manipulation of its predictors. Currently, a longitudinal study measuring cognitive development and its neural correlates and SES mediators as a function of either $333 or $20 monthly transfers to 1000 low–income mothers is being planned (Noble, 2017). Furthermore, some aspects of hypothesized mechanisms of SES–related disparities are under experimental control beyond intervention studies, such as investigating

parental effects on HPA axis activity (Hostinar et al., 2015b), environmental context effects on cognition, such as inducing poverty and resource restriction in games (Mani, Mullainathan, Shafir, & Zhao, 2013; Shah, Mullainathan, & Shafir, 2012), manipulating social status perception (Guinote, Cotzia, Sandhu, & Siwa, 2015), or inducing acute stress (Schwabe et al., 2007; Starcke & Brand, 2012). Although they cannot simulate cumulative experience, which of course is pivotal to theories of SES–related disparities, or genetic effects, experimental studies can deliver converging evidence for proximal environmental mechanisms.

Lack of genetic data

In this dissertation I have conceptualized cortisol dysregulation as a mediator of SES–cognition associations. However, this does not imply that cortisol dysregulation is exclusively, or predominantly, a mediator of environmental, and not genetic, effects. Twin studies suggest that many effects ascribed to SES are genetically confounded (Ericsson et al., 2017; Krapohl & Plomin, 2016; Polderman et al., 2015; Spinath & Bleidorn, 2017). Thus, it is likely that our correlational results partly derive from genetic effects influencing children's cortisol secretion, hippocampus volume, memory functioning, parent stress, and income (*Papers I & II*). For instance, the moderation effect of cognitive control reported in *Paper II* could reflect a genetic effect of environmental susceptibility, plasticity for learning, and intellectual interest (Blair & Raver, 2012; Tucker–Drob & Harden, 2012b). Therefore, it is currently not established whether cortisol is, in fact, a mechanism of SES–related stress on cognitive development (see Figure 4a), or, alternatively or additionally an intermediate biological mechanism (endophenotype) of genetic effects on child cognition (see Figure 4b).

It is less clear, but not implausible, how genetic effects would account for cross–lagged income–to–cognition couplings seen in poor children (*Paper I*). Furthermore, longitudinal mediation studies (Hackman et al., 2015), adoption studies (Capron & Duyme, 1989; Kendler et al., 2015; van IJzendoorn et al., 2005), (quasi–) experimental and intervention research (Costello et al., 2003; Duncan et al., 1998; Heckman, 2006), and genetically informed studies (Tucker-Drob & Briley, 2014; Tucker-Drob et al., 2013; Tucker-Drob & Harden, 2012b) provide evidence that environments along SES strata are also likely to play a causal role in childhood cognitive development that may be most pronounced, but not limited to, poverty and early childhood. Critically, very few twin studies are socioeconomically representative and tend to underrepresent twins living in low SES and poverty. For example, childhood SES is indicated by parental occupation that does not indicate poverty in Ericsson et al. (2017). Furthermore, the prenatal environment, which is more closely shared by monozygotic than dizygotic twins as they can share a placenta and amniotic sac, may to some extent affect genetically–mediated effects and overestimate heritability estimates (McGue, 1997). Thus, there is evidence that SES–related individual differences in cognition, and perhaps cortisol secretion, is substantially driven by genetic effects, but is also modifiable by environmental input.

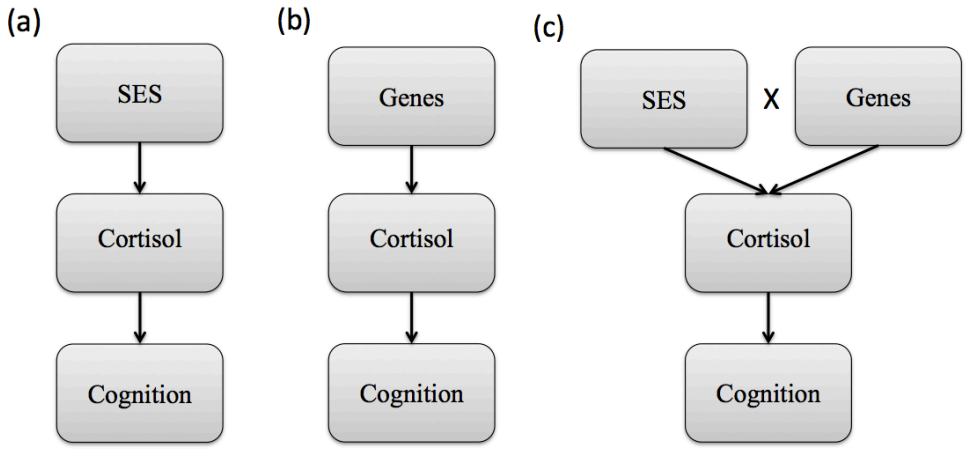

Figure 4. Three conceptual models for the role of cortisol (a) as a mediator of the effects of SES–related stress on children's executive function, (b) as an endophenotype of genetic influences, or (c) as a mediator of SES moderation effects on genes.

Emerging research also proposes that SES *interacts* with genetic effects in predicting child cognition (Harden, Turkheimer, & Loehlin, 2007; Rhemtulla & Tucker–Drob, 2012; Tucker–Drob & Bates, 2016), although such effects may be limited to the US amongst reported countries (Tucker–Drob & Bates, 2016). Genes explain more of the variance in cognition and brain structure in high–SES individuals than in low–SES individuals (Chiang et al., 2011; Tucker-Drob & Bates, 2016; Tucker-Drob & Harden, 2012b; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). In low SES, cognitive ability is almost entirely predicted by environmental factors, whereas high–SES environments facilitate children to select learning experiences that better match their genetically influenced individual differences in interest (Tucker–Drob & Harden, 2012b). This implies that low–SES environments may suppress gene expression on child cognition. In contrast, enriched environments allow for more dissimilarity in the experiences organisms make within the same environment, even when they are genetically identical (Freund et al., 2013). An SES x gene interaction could partially explain results in *Paper I*, such that the SES–cognition correlation in never poor children derives from a genetic effect, whereas income–related environments could influence poor children's cognition.

Animal research suggests that one mechanism for genetic suppression may be GC response to stress that changes the expression of genes involved in neural development, thereby leading to poorer memory (Meaney, 2010). Given aberrant cortisol secretion in low–SES children, it is conceivable that cortisol is the underlying mechanism that suppresses gene expression related to neural growth in low SES (see Figure 4c). This could explain the SES x CSR interaction observed in *Paper III,* such that cortisol functions as a mediator of low–SES suppression effects on gene expression. However, the aforementioned cross–national meta–analysis suggests that SES x gene effects on intelligence are not found in European samples combined across several countries (and including one German sample)

(Tucker–Drob & Bates, 2016). Furthermore, the presence or absence of SES x gene effects may be dependent on sample age, although evidence is currently inconsistent (Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011, but seeTucker-Drob & Bates, 2016). Therefore, exploring SES x gene effects awaits further empirical scrutiny cross–nationally. Behavior genetic and genome–wide association research provide elegant methods to elucidate the interplay of SES, cortisol secretion, and (epi–)genetic effects in explaining or eliciting SES disparities in cognition (Spinath & Bleidorn, 2017; Swartz, Hariri, & Williamson, 2017).

5.3 Conclusions and implications

The goal of the present dissertation was to investigate longitudinal dynamics of SES and cognition and test whether stress mechanisms are involved in cognitive disparities. To achieve this goal, *Paper I* drew from a large longitudinal dataset, suggesting that poor children's cognitive performance continues to be predicted by income gains and losses throughout later childhood. This highlights the need to look at income changes by treating income as a variable system and to allow for moderation of poverty. Exogenous income increase interventions in later childhood may therefore improve within–person cognitive development even when between–person comparisons to controls are modest (e.g., Dahl & Lochner, 2012). Furthermore, we showed that children's cognition in later childhood influences their parents' earning ability, indicating previously neglected bidirectional family dynamics. Correspondingly, early childhood educational intervention programs with the largest effects involve family dynamics (Dodge, Bai, Ladd, & Muschkin, 2017; Heckman, 2006; Sanders & Mazzucchelli, 2013). To illuminate the bidirectional relationship of income and cognition, longitudinal studies should include comprehensive measurement of many potential environmental and genetic mediators at each measurement occasion, which will require thoughtful statistical analysis and well–powered designs (Brandmaier, Prindle, McArdle, & Lindenberger, 2016).

Second, this dissertation offers methodologically reliable and valid evidence that lower income and higher parenting stress is associated with hypocortisolism in middle childhood (*Papers II & III*). I highlight the value of applying latent–change SEMs (*Papers I & III*) to the study of longitudinal development and dynamic cortisol reactivity. By treating cognition as the moderator, *Paper II* added the insight that the association of parenting stress and children's cortisol diurnal levels were buffered if the latter had higher cognitive control. Further, *Paper III* showed that CSR hyporeactivity was associated with lower memory among lower–income children, who also had smaller hippocampal volumes. Yet, we find no evidence that these smaller hippocampal volumes are associated with SES–related disparities in associative memory, which points towards hippocampal plasticity for learning as the potential mechanism for future investigations (Gray et al., 2013). These findings provide initial empirical evidence that low–SES is related to attenuated HPA axis activity in children, such that hyporeactivity to stress is negatively associated with memory functioning. Notably, evidence of a stress mechanism presented here, refers to evidence of cortisol dysregulation associated with lower SES or higher stress exposure. It does not imply environmental– or genetically–mediated causation, as the studies lack genetic and longitudinal data.

Although children are particularly vulnerable to effects of stress (Shonkoff et al., 2012), they are also more malleable in response to intervention, profoundly so in the preschool age (Heckman, 2006). Future interventions should test whether increasing income gains, lessening family stress exposure, and improving acute stress coping in lower SES children could attenuate cognitive disparities. Cortisol levels are also modifiable through psychosocial interventions (Slopen,

McLaughlin, & Shonkoff, 2014) and targeting cognitive control may improve outcomes. Since HPA axis dysregulation is known to be involved in health and psychiatric outcomes (Danese & McEwen, 2012), these results may also have implications for child outcomes other than cognition.

This research leads to a number of further questions to explore: Is hypocortisolism the outcome of SES–related stress or a vulnerability factor? Is the association of cortisol hyporeactivity to stress with associative memory driven by aberrant hippocampal function? Is cortisol a mechanism for the effects of chronic stress or, additionally a physiological endophenotype that mediates genetic influences on child cognition? Modeling bidirectional within–person dynamics of longitudinal data provide an excellent tool to clarify whether stress precedes cortisol dysregulation and whether this leads to cognitive deficits or vice versa. In addition, experimental studies that assess the impact of changing SES or associated mechanisms on cognitive development are needed to determine causal links. Techniques of behavioral genetics and newly developed genome–wide association studies offer much promise for disentangling environmental and genetic causes of cognitive disparities and exploring their interactions.

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